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"ALCOHOL" AMBLYOPIA, PELLAGRA, POLYNEURITIS

REPORT OF TEN CASES

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That so-called alcoholic polyneuritis and the alcoholic type of pellagra are deficiency diseases is now commonly accepted. A review of all the many studies which tend to prove this is not warranted here, but the evidence may be briefly abstracted as follows:

Shattuck [1] in 1928 and Minot [2] in 1929 suggested that avitaminosis might be an important factor in the etiology of alcoholic polyneuritis, because of the similarity of this disease to beriberi.

Wechsler [3] in 1930 and 1933 reported cases of polyneuritis in patients taking an inadequate diet. The polyneuritis was greatly benefited by the proper diet.

Minot, Straus and Cobb [4] found that almost all their patients with this condition had been on a deficient diet. They stated that "dietary deficiency, probably especially the lack of vitamin B_1, plays an important rôle in the production of 'alcoholic' neuritis."

Spies and De Wolf [5] allowed ten hospitalized patients with pellagra (the alcoholic type) to consume from 600 to 900 cc. of whisky daily, provided they took a well balanced diet plus 75 Gm. of autoclaved yeast daily. The patients recovered from pellagra very well despite the intake of alcohol.

Straus,[6] in a convincing study, permitted ten patients with alcoholic polyneuritis to continue their customary intake of spirituous liquor (from 1 pint to 1 quart [473 to 946 cc.] daily) but fed them a well balanced

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1. Shattuck, G. C.: Am. J. Trop. Med. **8**:539, 1928.
2. Minot, G. R.: Ann. Int. Med. **3**:216, 1929.
3. Wechsler, I. S.: M. J. & Rec. **131**:441, 1930; Etiology of Polyneuritis, Arch. Neurol. & Psychiat. **29**:813 (April) 1933.
4. Minot, G. R.; Straus, M. B., and Cobb, S.: New England J. Med. **208**: 1244, 1933.
5. Spies, T. D., and De Wolf, H. F.: Am. J. M. Sc. **186**:512, 1933.
6. Straus, M. B.: Am. J. M. Sc. **189**:378, 1935.

diet supplemented with yeast or its products, vitamin B concentrates and liver extract given parenterally. The rapidity of recovery was as rapid and the degree of recovery as complete in the patients who drank this large quantity of whisky as in the control group who received no alcohol.

Cowgill [7] calculated what appeared to be the vitamin B requirement of man. Jolliffe, Colbert and Joffe,[8] using this calculation, found that in a series of forty-two alcohol addicts, twenty-six of whom had polyneuritis, all twenty-six had an estimated inadequate intake of vitamin B. The alcohol addicts who did not have polyneuritis had an estimated adequate intake of vitamin B.

Wechsler, Jervis and Potts [9] kept monkeys and cats which were on a normal diet intoxicated with alcohol for as long as two years. No special histologic changes were observed in the nervous system. But in another series of monkeys fed a diet deficient in vitamin B and kept intoxicated, marked and widespread lesions of the central nervous system were observed.

It is my purpose in this paper to report ten cases of the clinical syndrome variously called tobacco-alcohol, tobacco or alcohol amblyopia, which occurred in patients with the alcoholic type of pellagra or polyneuritis. Clinical entities such as pellagra and nutritional polyneuritis are relatively rare, but symptoms resulting from a deficiency in specific nutritional factors occur frequently.[10] For many years various clinicians [11] have suggested that faulty nutrition was an important factor in the production of this ocular condition; at present physicians are gradually arriving closer to an idea of what this inclusive term "faulty nutrition" means.

REPORT OF CASES

CASE 1.—G. W., a 35 year old unemployed clerk, was first seen by me in the Vanderbilt Clinic on March 19, 1935. He said that he had been drinking between 1 and 3 pints of whisky daily for many months and that nine days previously, on awaking from a three day drinking "spree" he first noticed poor vision. He smoked one package of cigarets daily. Vision in the right eye was 20/70—, and in the left, counting fingers at 2 feet (60 cm). Vision in the left eye had always been

7. Cowgill, G. R.: The Vitamin B Requirement of Man, New Haven, Conn., Yale University Press, 1934.

8. Jolliffe, N.; Colbert, C. N., and Joffe, P. M.: Am. J. M. Sc. **191**:515, 1936.

9. Wechsler, I. S.; Jervis, G. A., and Potts, H. D.: Bull. Neurol. Inst. New York **5**:453, 1936.

10. Haden, R. L.: Multiple Specific Nutritional Deficiency Disease in the Adult, J. A. M. A. **106**:261 (Jan. 25) 1936.

11. Mackenzie, William: A Practical Treatise on the Diseases of the Eye, Philadelphia, Blanchard & Lea, 1855, p. 987. Traquair, H. M.: Edinburgh M. J. **42**:153 (Aug.) 1935. Holth, S.: Acta ophth. **5**:195, 1927. Letchworth, T. W.: Clin. J. **61**:137, 1932. Carroll, F. D.: Analysis of Fifty-Five Cases of Tobacco-Alcohol Amblyopia, Arch. Ophth. **14**:421 (Sept.) 1935.

poor, owing to amblyopia ex anopsia associated with a central scotoma; the vision and central field in this eye have remained unchanged, so they will not be mentioned again in this report. The eyes showed a horizontal nystagmus on looking to either side and a vertical nystagmus on looking up; the right fundus showed one striate hemorrhage, and the left fundus had three such hemorrhages. The patient had been vomiting severely, and the hemorrhages were possibly due to that. He had been cooking for himself, and his diet was very inadequate. Diarrhea had been present for several weeks.

In three days the central scotoma for colors in the right eye cleared up entirely without any treatment, and vision became 20/20 in the right eye. Two weeks later, however, he returned to the clinic because of blurred vision. He had continued to drink heavily. Vision in the right eye amounted to counting fingers at 3 feet (91.4 cm.), and there was a large centrocecal scotoma. He was admitted to the Institute of Ophthalmology. A neurologist found diffuse neurologic lesions, which included partial motor weakness of the left fifth nerve, a decrease in hearing, hypo-active reflexes in the upper extremities and hyperactive reflexes in the lower extremities. He made a diagnosis of multiple sclerosis, which, as will be seen, proved to be incorrect.

Visual and auditory hallucinations developed, and the patient began to show marked retardation of cerebration. The diarrhea became worse. At this time dry, scaly, slightly pigmented lesions were noted on the hands and feet, and pellagra was diagnosed. Because of psychic disturbances the patient was transferred to the New York State Psychiatric Institute. He was placed on an antipellagra regimen, which consisted of a diet high in vitamin B_1 and B_2, plus liver extract injected intramuscularly. The diarrhea became so bad that a rectal tube had to be left in place; the patient was expected to die. Slowly, however, his physical condition improved; his visual acuity and fields remained essentially unchanged until July 11, when vision in the right eye had improved from counting fingers at 3 feet to 20/40+. There was still a centrocecal scotoma for colors. Two months later vision in the right eye was 20/25. Now, sixteen months since the onset of the amblyopia, vision in the right eye is 20/20; there is a small scotoma for colors between the blindspot and the area of fixation; the temporal part of both disks shows moderate pallor. The patient has slight residual mental impairment, but his physical condition is excellent.

CASE 2.—E. F., a 32 year old Negro postoffice clerk, was seen by me at the Vanderbilt Clinic on May 13, 1935. His vision had been failing for six months; he habitually drank 1 pint of whisky and smoked 4 or 5 cigars daily. Twenty-four hours before he came to the clinic he had three visual hallucinations: He saw two cars collide, saw fire spring out of the street and saw rain falling while the sun was shining. He realized that these were "visions." Vision in the right eye was 18/200 and in the left 10/200; the disks appeared normal; there was a large centrocecal scotoma with normal peripheral fields (fig. 1). Recalling my experience in case 1, I inspected the patient's skin immediately and found the lesions shown in figures 2 to 5. These were the typical cutaneous lesions of pellagra.

In two days vision in both eyes had decreased to counting fingers at 3 feet. The patient was hospitalized and given antipellagra treatment. The dermatitis disappeared in three weeks. Polyneuritis developed while the patient was being treated; this is a common occurrence in pellagra, and it may last weeks or months. A mild stomatitis quickly improved. Vision gradually improved to 20/40+ in both eyes in the course of thirty-five days and now, twelve months later, is 20/20— in both eyes.

Case 3.—C. B., a 25 year old Negress, a patient in the dermatology service of the Bellevue Hospital, had the cutaneous lesions of pellagra on the hands, elbow and vulva. She had been drinking 1 quart of whisky and smoking a few cigarets daily for many months. Her diet was very inadequate. Examination of the eyes showed vision of counting fingers at 5 feet (152 cm.) in both eyes. The disks showed marked pallor in the temporal portion. Examination of the fields demonstrated a large centrocecal scotoma in both eyes.

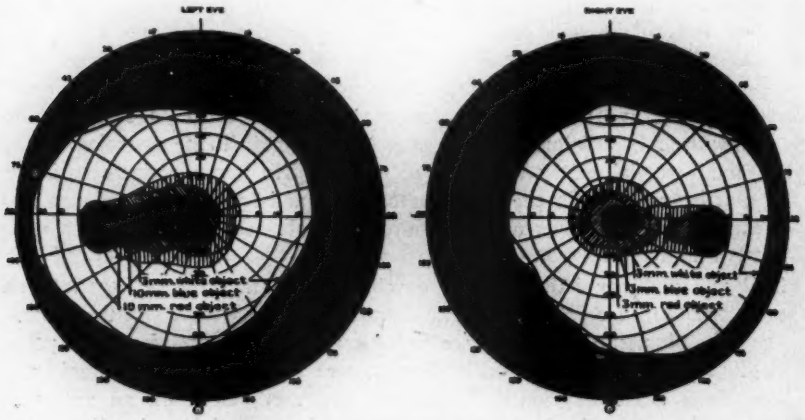

Fig. 1 (case 2).—Visual fields, taken after the patient was hospitalized. Vision was counting fingers at 3 feet. The fields for 3 mm. white, blue and red test objects at 1 meter are shown.

Fig. 2 (case 2).—Lesions on the wrists.

Case 4.—W. R., a 44 year old taxi driver, a chronic alcohol addict, was seen at the Bellevue Psychiatric Hospital. He was accustomed to drinking 2 or 3 pints of whisky and smoking one package of cigarets daily. The diagnosis of his condition was pellagra, polyneuritis and Korsakoff's psychosis. The left eye had a mature traumatic cataract and faulty light projection. Vision in the right eye was counting fingers at 3 feet (91.4 cm.). The temporal portion of the disk showed marked pallor compatible with atrophy. Satisfactory visual fields were not obtained until the psychosis was cured. A typical centrocecal scotoma with

normal peripheral fields was then found. The dermatitis cleared up in one month, the polyneuritis, in two months; vision in the right eye has remained at 20/200 for six months.

CASE 5.—N. M. was admitted to the Bellevue Psychiatric Hospital on Dec. 12, 1935, in a stuporous condition. He was disoriented and showed mental impairment. There was brawny edema of the legs and thighs. He had the classic cutaneous lesions of pellagra, as well as mild polyneuritis. The history, which was obtained later, revealed that he was an unemployed janitor who had been drinking at least 1 quart of whisky daily since losing his job nine months previously. Often he

Fig. 3 (case 2).—Lesions on the dorsa of the hands.

drank 2 quarts of whisky daily. He had smoked from ten to twelve cigarets a day for a long time. His caloric intake was largely derived from alcohol.

When I first examined the patient three weeks after his admission to the hospital vision in the right eye was 10/200 and in the left 8/200; the temporal part of the disks showed moderate pallor, and the fields showed centrocecal defects. On Jan. 7, 1936, he was transferred to the Institute of Ophthalmology, where the proper dietary treatment was continued. The cutaneous lesions gradually improved; the pain in the feet, associated with the polyneuritis, first became worse and then was completely cured. Vision in the left eye had never been very good; retinoscopy revealed a high degree of astigmatism. However, in the course of three months vision in the left eye improved to 20/80— with correction, and that in the right eye, to 20/40—. Treatment consisted, in part, of a high

vitamin diet plus brewers' yeast, wheat germ (Embo) and Vegex by mouth, as well as Vitamin B_1 concentrate and liver extract given intramuscularly.[12]

Case 6.—A. P., a 31 year old Negro, was examined at the Bellevue Psychiatric Hospital. The diagnosis of his condition was pellagra, polyneuritis and alcoholic encephalopathy. His brother said that he had been drinking more than 1 quart of gin daily for a long time, probably several years. He usually smoked a few cigarets daily. The patient's physical and mental condition improved, and three weeks after admission my examination of the eyes showed vision of 15/200 in the right eye and 20/40 in the left, slight pallor of the temporal part of both optic

Fig. 4 (case 2).—Lesions on the extensor surfaces of the elbows.

disks and a centrocecal scotoma in both eyes. Two weeks later vision in the right eye was 20/50, and in the left, 20/30.

Case 7.—A 35 year old white patient with pellagra associated with alcoholism was seen by me in 1934 in the dermatology ward at the Massachusetts General Hospital, Boston. He had been drinking from 1 to 2 pints of whisky and smoking one package of cigarets daily. Vision was 20/200 in both eyes, and he had the typical central field defects of alcohol amblyopia.

Case 8.—A. R., a 34 year old white woman, was admitted to the Bellevue Psychiatric Hospital on July 25, 1935. She was confused and disoriented and

12. The brewers' yeast used was furnished by Anheuser-Busch, Inc.; the wheat germ, by the General Mills, Inc., and the liver extract by Eli Lilly & Co.

had hallucinations. The history as obtained from her brother and confirmed several weeks later by the patient was that she had been drinking heavily for several years and during the past year had been consuming approximately 1 quart of whisky daily. She smoked one package of cigarets a day. Two months previously she had been at another hospital because of alcoholic neuritis. The diagnosis was Korsakoff's psychosis and alcoholic polyneuritis. She had no cutaneous lesions. One month later the knee and ankle reflexes were still absent, and there were hyperesthesia of the soles, absence of vibratory sense in the toes and a subjective feeling of numbness in the fingers, toes and soles, as well as some impairment of memory for recent events. My examination of the eyes on October 14 showed: vision of 20/30 in both eyes, a centrocecal scotoma in both eyes for 1 mm. red and green test objects at a distance of 19 cm. and slight nystagmus on lateral gaze. The patient stated that her vision had greatly improved since she was in the hospital. Two weeks later the scotomas were definitely smaller and occupied an area between the blindspot and the fixation point in each field.

Fig. 5 (case 2).—Lesions between the buttocks.

CASE 9.—C. M., a middle-aged white man, was admitted to the Bellevue Phychiatric Hospital on June 11, 1936. The diagnosis was chronic alcoholism, polyneuritis, nutritional edema and pellagra. He said that he had been drinking from 1 to 4 pints of corn whisky daily and smoking not more than one package of cigarets a day for many months. He had been existing almost entirely on white bread, soup and whisky. The cutaneous lesions were of the type found in pellagra and were located on the hands, forearms, feet and legs. There was pitting edema of the ankles and feet. The under-surface of the tongue was red, and the margins were smooth. The patient stated that he had had similar cutaneous lesions a year previously, when he was drinking heavily, and that the cutaneous condition disappeared when he decreased his intake of alcohol. He had noticed some impairment in vision recently. Examination of the eyes by me on June 15 revealed vision of 20/40 in both eyes, marked horizontal nystagmus on lateral gaze, moderate pallor of the temporal part of both disks and a centrocecal scotoma for 1 and 3 mm. red test objects at a distance of 19 cm. in both eyes.

CASE 10.—M. C., a 30 year old white man, seen at the Bellevue Psychiatric Hospital, had the following interesting history: He said that he had been a

heavy drinker for fifteen years and that he consumed from 1 pint to 3 quarts of whisky daily. He smoked from one to two packages of cigarets a day. He was not disturbed about his amblyopia because he had experienced several similar attacks after periods of unusually excessive drinking. He said the blurred vision usually disappeared in from one to six weeks if he moderated his intake of alcohol. The patient had the stomatitis, diarrhea, dermatitis and polyneuritis so often seen in persons with pellagra. On Oct. 19, 1936, the vision in each eye was 20/200. The patient was given an antipellagra diet, and one week later his general condition as well as the condition of his eyes was greatly improved. Vision was then 20/50 in the right eye and 20/100 in the left eye. The centrocecal scotomas were smaller and less dense.

SUMMARY

A short abstract is presented of the evidence for the conclusion that the alcoholic type of polyneuritis and the alcoholic type of pellagra are deficiency diseases associated with the lack of the vitamin B (B_1, B_2, etc.) fraction. Ten cases of "alcohol" amblyopia are reported which occurred in patients who had pellagra or polyneuritis or both. All these patients smoked in moderation, and all but one used only cigarets. But they were all heavy drinkers: They consumed between 1 and 3 quarts of alcoholic liquor daily. Most of them were dipsomaniacs. In every case the diet had been inadequate.

THE TENNENT CHAIR AND THE TENNENT MEMORIAL INSTITUTE OF OPHTHALMOLOGY, GLASGOW

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Clinical ophthalmology has been taught in Glasgow since Dr. William MacKenzie and his colleague, Dr. George Monteith, in 1824, were instrumental in founding the Glasgow Eye Infirmary, and a lectureship in the subject was instituted by Glasgow University in 1828. This lectureship was held in succession by Dr. William MacKenzie (1828-1868), Dr. George Rainy (1868-1869) and Dr. Thomas Reid (1869-1900) and after an interval of eleven years by Dr. A. Maitland Ramsay (1911-1920) and myself (1920-1935).

The lecturer occupied a somewhat anomalous position. In the first place, the university had no special lecture rooms or laboratories for ophthalmology; second, ophthalmology, for purposes of graduation, was essentially a clinical subject, and systematic lectures formed no necessary part of the students' course; third, qualifying courses in Ophthalmology could be conducted not only by the university lecturer but also by the other surgeons of the special hospitals (the Eye Infirmary and the Ophthalmic Institution); and, fourth, postgraduate teaching of ophthalmology was almost purely clinical and was also conducted in these special hospitals.

On the clinical side, therefore, with some nine or ten recognized teachers and with the abundant material of the special hospitals (with some 50,000 new outpatients per annum), ophthalmology was and is fully provided for, and clinical teaching has always reached a high standard, although the minimum length of the qualifying course (one term of three months) is regrettably short.

A generous bequest by Dr. Gavin P. Tennent, who died in 1913, placed ophthalmology on an entirely new footing in Glasgow, for it enabled the university to found a chair of ophthalmology and at the same time provided funds for the building of an institute of ophthalmology, which is attached to the Western Infirmary and provides both for the care of patients suffering from diseases of the eye and for teaching and research in ophthalmology.

Dr. Tennent was a member of the medical staff of the Western Infirmary from 1874 till 1897, when, on his retirement, the managers recognized his services to the institution by appointing him the first honorary consulting physician. On the occasion of the inauguration of the new institute Dr. A. Maitland Ramsay, who had a long and intimate association with Tennent, paid a warm tribute to his qualities as physician and friend, his courtesy and friendliness, his sincerity and lack of affectation, his human sympathy and understanding and his sound training, accumulated experience and well developed judgment, which justified the implicit trust as well as the affection of his patients.

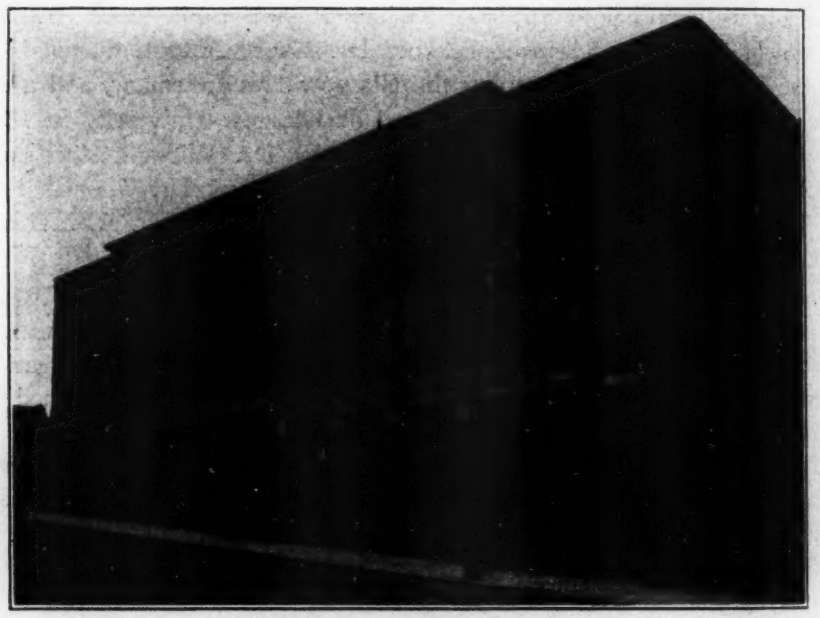

Fig. 1.—The Tennent Memorial Institute.

Although Tennent died in 1913 and the foundation of the chair of ophthalmology dates from 1917, circumstances, arising for the most part out of the World War, delayed the consummation of the memorial scheme, but in May 1935 I was appointed to the chair; in the early part of this year the building of the institute was completed, and its formal inauguration took place on April 9.

The building faces west, and on the ground floor a corridor runs from west to east and, connecting with the main building of the Western Infirmary, divides the outpatient department from the rooms for teaching and research. The outpatient department consists of a patients' waiting room, a consulting room, a refraction room, a casualty room

and a dark room. The last provides ten roomy cubicles for retinoscopy and ophthalmoscopic work, while four large cubicles contain the slit lamp, keratometer, binocular ophthalmoscope and red-free lamp. In addition to the electric mains providing alternating current of 250 volts low tension tapping of 3, 6, 8 and 12 volts are provided for special instruments.

In the other part of the ground floor are the staff room, the secretary's office, the library, the director's office, the laboratory, the research laboratory and the lecture room, the last of which can be divided into two rooms by folding partitions.

On the first floor the operating theater and its annexes are situated above the outpatient department. The south portion of this floor is

Fig. 2.—Two views of a ward.

occupied by the wards for male patients. These consist of a small ward of four beds, five single bed cubicles and a single room with a bed. The apartments are isolated from each other by glass partitions, and, if necessary for examination or other purposes, any one of the cubicles can be darkened by curtains. Normally the beds can all be kept under observation from the nurse's station. A balcony runs along the south aspect, and each patient, either in bed or otherwise, has access to the balcony. On this floor the other apartments are the necessary bathrooms, the ward kitchen, etc., a patients' day room and a clinical laboratory.

On the second floor the accommodation for female patients repeats the design of the wards and other apartments on the first floor. There is also a second clinical laboratory, and above the operating theater there is a studio with other apartments for clinical photography, photomicrography and drawing.

The Tennent Memorial Institute thus provides a department for patients with diseases of the eye, for both inpatients and outpatients, for the western infirmary, and, at the same time, a teaching and research department for the university; and the Tennent Professor of Ophthalmology is, at the same time, visiting ophthalmic surgeon to the Western Infirmary. The chair of ophthalmology is not a full time appointment, and the professor and his assistants are allowed to engage in private practice. While a limited number of undergraduates are allowed, the main duty of the professor is the promotion of "higher study and research" in ophthalmology. Postgraduate work will therefore receive special attention and will be adapted to meet the requirements of advanced students, general practitioners in need of "refresher" courses, candidates for diplomas in ophthalmology and others.

In April 1936, in pursuance of an exchange of lectures between the University of Glasgow and the University of Basel, Professor A. Brückner, of Basel, Switzerland, delivered in the new institute six lectures dealing with certain aspects of light and color vision, Professor Ballantyne having given a course of lectures in Basel in June 1935. In September a short course of lecture demonstrations will be given in the Tennent Institute, dealing with ocular manifestations of cardiovascular disease, tuberculosis, venereal diseases, toxic states and injuries to the head.

MONOCYTIC CHLOROMA

(RETICULOCYSTOBLASTOMA WITH MONOCYTIC LEUKEMIA)

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The citation of rare cases is of value only so far as it makes for more exact diagnosis, which in turn tends to make the treatment of disease less empirical. The purpose of this paper is to report a case in which there were a blood picture of monocytic leukemia and pathologic involvement of the reticulo-endothelial system. For this clinical picture the title of monocytic chloroma is suggested. A brief review of the literature on chloroma, monocytic leukemia and the reticulo-endothelial system is also presented.

REPORT OF CASES

History.—F. C., a white American laborer 55 years of age, was brought to the Eye Clinic of the University Hospital on Nov. 26, 1934. He complained of protrusion of the left eye associated with swelling of the lids, diminution of visual acuity, a feeling of fatigue in both eyes and a bulging red mass at the inner angle of the left eye between the margins of the lids.

In the latter part of September the patient had had symptoms of asthenopia, noted especially for near vision. Late in October he first noted swelling of the eyelids on the left and some prominence of the globe. This gradually progressed, without pain or discomfort. Early in November the patient discovered that he could read only headlines of newspapers and that he had difficulty making out familiar objects across the room. Previously his vision had been unimpaired. An optometrist fitted him with glasses, which relieved the ocular fatigue previously noted but did not improve visual acuity.

Ten days prior to hospitalization the patient became aware of a red bulging mass of tissue at the inner angle of the left eye in the palpebral fissure. This mass increased in size until it filled the entire lower half of the fissure. There were some associated tearing and at times gross discharge.

The patient was referred to the university hospital by his ophthalmologist for further study.

The family history and the marital history were unimportant as regards the illness. The patient had been a common laborer and had worked until nine days before hospitalization. The past history was not significant except that the patient had had rheumatism in the left leg for two years. The history according to systems was without import except for a purulent painless discharge from the

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right ear of two weeks' duration one year previously, with a recurrence bilaterally for two months prior to entrance. This was associated with diminution of hearing. The patient's mouth had been sore since the onset of the illness for which he was admitted. He had several carious teeth. There had been a recent loss of weight of approximately 15 pounds (6.8 Kg.).

Physical Examination.—The patient was a well developed, moderately well nourished swarthy white man about 55 years of age and about 6 feet (183 cm.) tall. He did not appear to be acutely ill. Attention was at once drawn to the left eye, which was proptosed, and to the eyelids, which were edematous.

Examination of Eyes: External.—Vision in the right eye was 6/15 and in the left 6/30. With correction vision in the right eye was 6/6 and in the left 6/30. Tension on tactile pressure was normal in both eyes. The exophthalmometer reading was 21 mm. for the right eye and 27.5 mm. for the left.

The right eye could be elevated only slightly above the midline. Depression was fairly good. Abduction and adduction were definitely limited. The orbital margins could be palpated and were normal. The palpebral conjunctiva showed subacute injection. There was definite chemosis of the conjunctiva below and temporally, though it did not bulge above the margins of the lids into the palpebral fissure. The globe was proptosed directly forward.

Fig. 1.—*A,* anteroposterior view of the patient on Nov. 11, 1934, showing exophthalmos and conjunctival chemosis of the left eye. *B,* lateral view of the patient.

The extra-ocular movements of the left eye were definitely and considerably limited in all directions. The conjunctiva below the cornea was very chemotic. It protruded between the lids and was dry on its exposed surface. The lower lid was pushed down by the edematous conjunctiva. The conjunctiva above was moderately chemotic and subacutely injected. There was moderate edema of the upper lid. A rather hard tumor could be palpated in the orbit below and temporal to the globe. The globe was proptosed directly forward.

Examination of the Fundi.—There were early lenticular opacities in the lower nasal periphery of the right eye. The media were somewhat hazy. The papilla was blurred and showed aproximately 1.5 diopters of papilledema, which was fairly well confined to the nerve head. There was a minute hemorrhage on the disk above. The retinal veins showed engorgement and tortuosity.

The media of the left eye were hazy, somewhat obscuring the finer details of the fundus. There was approximately 3 diopters of papilledema which was fairly well confined to the nerve head. There were engorgement and tortuosity of the retinal veins, which were more marked than in the right eye.

Examination of the visual fields showed on the right slight concentric contraction for form and color. The blindspot was approximately one and one-half times the normal size. On the left there was considerable concentric contraction for form and color. The blindspot was about three times normal size.

Summary of Findings.—The summary of the findings of the complete physical and ocular examination was as follows: An orbital tumor in the left eye; papilledema in both eyes; exophthalmos in both eyes; depressed vision in the left eye; limitation of extra-ocular movements in both eyes; concentric contraction of the visual field, with enlarged blindspot, in both eyes; bilateral diminution of hearing; bilateral external otitis with a purulent exudate; deviation of the nasal septum; dental caries and fragments of roots of teeth; gingivitis; fine moist râles at the base of the left lung; enlargement of the spleen; palpable lymph nodes in the cervical, axillary and inguinal regions; external hemorrhoidal tags, and tenderness of the prostate.

Progress.—The patient was admitted to the ward of the ophthalmologic service on November 26. Several extractions of teeth were done by oral surgical measures without complication. A roentgenogram of the skull and of the paranasal sinuses showed slight thickening of the mucosa of the left frontal sinus and moderate hyperplasia of the antral mucosa. There were an orbital tumor of the left eye, with increase of density of soft tissue; enlargement of the orbit, and apparent invasion of the tumor through the medial wall to involve the ethmoid cells. A diagnosis of malignant growth was considered. A roentgenogram of the lungs showed a fine granular appearance in the parenchyma suggesting early pneumonoconiosis.

A differential white cell count of the blood showed 43 per cent adult monocytes (table 2). This was checked by Dr. R. Isaacs, of the Simpson Memorial Institute, Ann Arbor. At this time petechiae developed over the abdomen, flanks and lower lumbar region. The patient was examined by Dr. R. Isaacs, who, on the basis of the blood picture and the clinical findings of orbital tumor, exophthalmos, petechiae, and enlargement of the spleen, left kidney and inguinal lymph nodes, made a diagnosis of the monocytic chloroma form of monocytic leukemia.

On December 3 a biopsy was done on tissue removed through a conjunctival approach in the lower cul-de-sac temporally. No solid tumor could be outlined, though the orbit was thoroughly probed. The tissue was edematous orbital fat and connective tissue. The pathologic report was as follows: "The tissue is neoplastic, related to a hematopoietic neoplasm such as reticulocystoblastoma or some form of myeloma or chloroma. The orbital fat was extensively infiltrated with large mononuclears (some of which suggest reticulocytes), lymphocytes and a few multinucleate giant cells. The condition resembles a group of conditions with retrobulbar infiltration resembling that in Hodgkin's disease. In this instance eosinophils are lacking."

On December 8 the patient became dizzy and unable to stand on going to the bathroom. At this time transient auricular fibrillation developed.

On December 10 a series of treatments by orbital and abdominal roentgen irradiation was initiated, as it was deemed that the neoplasm was radiosensitive. High voltage roentgen therapy was applied to a field 2 by 3 cm. in area, directly anterior and lateral to both orbits. The dose was 400 roentgens, measured in air, at a distance of 50 cm., with 200 kilovolts and 25 milliamperes, through a filter of 0.5 mm. of copper and 1 mm. of aluminum. With the same technic and with a dose of 300 roentgens, an area of 15 by 15 cm. over the liver and over

the spleen was irradiated. Table 1 shows the details of the course of therapy. There was no reaction following the orbital irradiations, but after each abdominal treatment there were nausea, anorexia, malaise and depression, lasting for twenty-four hours.

The exophthalmos remained approximately the same in the left eye as on admission, but gradually increased in the right eye until the eyes were about equally proptosed. The exophthalmometer reading on December 31 was 25 mm. for the right eye and 27 mm. for the left. By December 11 the orbital infiltration had increased so that there was complete loss of motility in the left eye and marked limitation of motility in the right eye. The edema extended progressively from the orbital region temporally and nasally; the edematous tissue overlapped the nasojugal fold on each side. This tissue on the right side was tender on palpation. The conjunctival chemosis increased gradually, especially on the right. On December 18 it extended over the lower lid of both eyes into the palpebral fissure and measured at its greatest vertical diameter 11 mm. in the right eye and 13 mm. in the left eye. At this time it was necessary to keep the exposed conjunctiva from drying and ulceration. It was covered with petrolatum, and the eyes were padded alternately for twenty-four hour periods. The cornea remained well covered by the upper lid on each side.

TABLE 1.—*Details of Course of Roentgen Therapy*

Date	Application	Dose, Roentgens	Area, Cm.
12/10/34	Lateral to right orbit	400	2 by 3
12/11/34	Anterior to orbits		
12/12/34	Anterior to orbits		
12/13/34	Lateral to left orbit		
12/14/34	Liver	300	15 by 15
12/15/34	Spleen		
12/17/34	Lateral to right orbit	400	2 by 3
12/18/34	Anterior to orbits		
12/19/34	Anterior to orbits		
12/20/34	Lateral to left orbit		

The picture of the fundus remained that of papilledema throughout. This did not measure more than 3 diopters at any time in either eye. Exudates appeared about the disk, gradually circumscribing them with numerous small flame-shaped hemorrhages on the margins of the disks and on the adjacent retina.

Vision gradually deteriorated, being 2/60 in the right eye and ability to count two fingers at 2 feet (60.9 cm.) in the left eye, on December 31.

Hearing diminished until on discharge the patient had difficulty hearing the normal spoken voice at 2 feet.

Following the biopsy a septic type of temperature developed and persisted throughout the remainder of hospitalization. It ranged from 100 to 104 F. each afternoon.

The petechiae previously noted involuted, and no new ones appeared until a subsequent hospitalization.

On December 31 the patient requested to be dismissed, as he believed that he could not get well. At home he became progressively worse and was admitted to the St. Mary's Hospital at Saginaw, Mich., for further roentgen therapy. He showed petechial hemorrhages over the entire trunk, and subsequently bilateral lobar pneumonia developed, which terminated fatally, death occurring on Feb. 2, 1935. Autopsy was done.

Laboratory Tests.—The routine Kahn test of the blood and examination of the urine and stool gave negative results. Table 2 shows the results of the studies of the blood.

Postmortem Examination.—This was performed on February 2 at 5:45 p. m. The skin showed numerous purpuric spots about 1 mm. in diameter scattered over the abdomen up to the costal margins, a few on the anterior wall of the chest and a number on the back. There were no external palpable glands, with the exception of slight enlargements in the inguinal region on both sides.

The eyes showed marked exophthalmos. The conjunctivae were everted, swollen and edematous. There was no evidence of pus formation. There was no discharge from the nose or the ears.

When the calvarium was removed, rather marked edema of the brain was noted. There were no hemorrhages. The cortical vessels were slightly promi-

Fig. 2.—Anteroposterior view of the patient on Dec. 20, showing exophthalmos, conjunctival chemosis and edema of the lids of both eyes.

Fig. 3.—Anteroposterior view of the patient on Dec. 28, showing exophthalmos, conjunctival chemosis and edema of the lids of both eyes.

nent. Cut sections of the cerebral hemispheres revealed no gross masses or hemorrhages. The pons orbitalis was rather eggshell in structure but was easily cut away, revealing on each side a rather large oval tumor which was deep yellow and occupied most of the orbital cavities. Each mass was attached to the posterior wall of the ocular bulb, the optic nerves bisecting the tissue anteroposteriorly in a diagonal direction. The mass in the left orbit measured 4.5 by 4.5 by 3 cm., the greatest diameter being in the anteoposterior direction. The right tumor measured 4 by 3.45 by 3 cm. The masses were firm and fibrous. Cut section revealed a shiny, fleshy tissue with radiating yellow bands about 6 mm. wide, surrounded by edematous-like areas. When the masses were removed from the orbit and detached from the eyeballs, the latter returned to their normal position. No other tumors were noted in this region. The sinuses and mastoids were carefully examined and observed to be clear.

TABLE 2.—*Results of Examination of the Blood*

Date	Hemoglobin, Percentage	Red Cells	White Cells	Differential Count, Percentage					Platelets	Comment*
				Polymorphonuclears	Eosinophils	Basophils	Lymphocytes	Monocytes		
11/27/34	65.6	4,100,000	15,500							
11/29/34	85.0	4,870,000	18,800	34.0	0	0.0	23.0	43.0	Normal number	
12/ 2/34	78.0	4,470,000	26,400	38.5	1	0.5	15.5	43.5	Normal number but some abnormal and large forms	Red cells normal in size and shape; no basophilic granules of polymorphonuclears †
12/ 4/34	75.0	3,610,000	12,400	53.0	0	0.0	6.5	41.0	Slightly increased in number	Red cells round, with rouleau formation; no basophilic granules of polymorphonuclears †
12/ 6/34	83.0	4,570,000	17,400	48.0	0	0.0	12.0	40.0		
12/13/34	82.0	3,646,000	12,400	60.0	1	0.0	19.5	20.0	.....	No basophilia; red cells round and well colored; reticulocytes not increased in number; size of red cells—7.5 microns, 51%; 7.5 microns, 40%; +7.5 microns, 9% †
12/19/34	77.0	.....	10,000	51.0	2	0.0	17.0	30.0		
12/31/34	61.0	3,760,000	12,350	32.0	1	1.0	4.0	62.0	Decreased in number; giant type present	Polychromasia; moderately severe secondary anemia (myelophthisic) from infiltration of bone marrow by malignant tissue; wiping out of lymphocytes due to roentgen therapy ‡

* The type cell varied from 11 to 21 microns in width and from 13.5 to 22.5 microns in height. Most of the cells measured, however, varied from 12 to 18 microns in width and from 15 to 21 microns in height.

† Studies of the blood were made by Dr. Isaacs, of the Simpson Memorial Institute, Ann Arbor.

‡ Examination of the blood was made by Dr. Goldhamer, of the Simpson Memorial Institute, Ann Arbor.

The right pleural cavity contained a small amount of cloudy fluid. The right lung was heavy and showed gray hepatization of the lower lobe and a mingling of red and gray hepatization in the upper lobe. Pus escaped from the smaller bronchi. The left lung was congested. There were small masses of firm yellow tissue in the posterior mediastinum. The heart was grossly normal.

The abdomen was distended, but there was no free fluid present. The stomach and intestines were normal. The mesenteric lymph glands were very large, some of them measuring 4.5 cm. in surface diameter and 2.5 cm. in thickness. Cut sections revealed a deep yellow surface, and the centers of some of them were rather dark green and showed some softening. The entire mesentery was thick and yellow. There were similar masses practically covering the pancreas but apparently not involving that organ. The liver was congested, but no gross masses were noted. The gallbladder and gall ducts were normal. The spleen was

Fig. 4.—Photograph of the patient taken on Jan. 22, 1935, showing exophthalmos, conjunctival chemosis and edema of the lids of both eyes, and petechial hemorrhages on the anterior part of the thorax.

pushed forward and to the right by a thick mass of the same type of yellow-streaked tissue noted elsewhere. This organ measured 14 by 7 by 5 cm. and was congested. No masses were noted on section.

The kidneys were covered with thick masses of dense yellow tissue which completely encompassed them. The capsule of both organs was 1 cm. thick, rather dense in consistency and streaked with the same yellow color noted elsewhere. The right kidney measured 12 by 6 by 5 cm.; the left, 11.5 by 6 by 5 cm. The cortical surface was fairly smooth. Cut sections showed the cortex to be indistinct in outline and about 7 mm. in width. The renal pelvis contained the same type of yellow tissue already described, which spread out in butterfly fashion, extending up to the pyramids and continuous with the midline. The mass in the right renal pelvis measured 5 cm. in length and 4 cm. in width. The adrenal glands were also covered with these masses.

The aorta was slightly thickened, and there were a few atheromatous patches in the intima.

Cultures of the lung revealed pneumococci of type IV.

Microscopic Observations.—Specimens were secured by Dr. O. W. Lohr, the pathologist, who performed the autopsy at the St. Mary's Hospital, Saginaw, Mich., and were examined and reported on by the pathology department of the University of Michigan. There were multiple foci of neoplastic cells in the subepicardial fat and beneath the endocardium. The lungs showed confluent fibropurulent pneumonia, and around many of the blood vessels and bronchi there were neoplastic infiltrations. The spleen showed focal areas of neoplastic infiltration. There was neoplastic infiltration of the pancreas, noted in the peripancreatic adipose tissue extending somewhat into the gland. There was no neoplastic

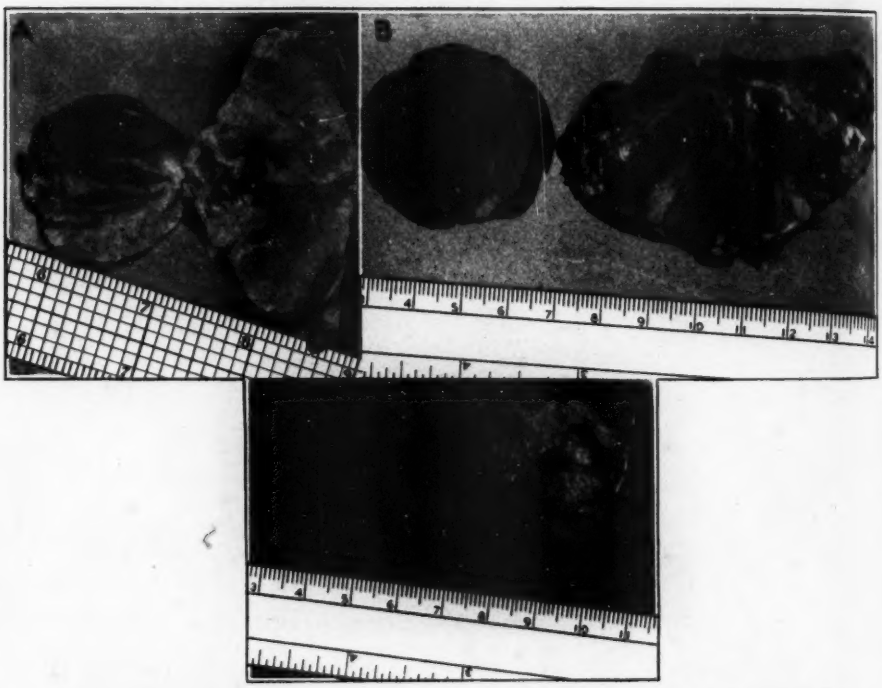

Fig. 5.—*A*, anteroposterior view of the orbital tumors. The left one is divided. *B*, vertical view of the right and the left tumor, respectively. The left one is divided. *C*, transverse view of the right and the left tumor, respectively.

involvement of the liver. The kidneys showed neoplastic infiltration around the renal vessels and extending into the kidney substance, and the retroperitoneal lymph nodes were surrounded by, and largely replaced with, neoplasm.

The neoplastic process, wherever observed, consisted chiefly of large polymorphous cells with pink-staining cytoplasm and a single eccentric spherical nucleus. Where it was fully established these cells lay in a connective tissue reticulum, giving the general appearance of granuloma. Particularly in the retroperitoneal region this neoplastic tissue contained many foam cells in which there were lipoids stainable by ordinary dyes for fat. In some regions there were numerous multinucleate giant cells.

Films of Blood Made on Glass Slides at Autopsy.—The determinations were made by Dr. Raphael Isaacs of the Simpson Memorial Institute, Ann Arbor. The white cell count did not appear to be increased. Polymorphonuclear neutrophils numbered 67 per cent; large lymphocytes, 6 per cent; small lymphocytes, 5 per cent, and monocytes, 22 per cent. The red cells showed considerable variation in size, and an occasional distorted form was present. The platelets were somewhat decreased in number. The blood did not appear to be grossly abnormal except for the presence of young and mature monocytes.

Films of the Bone Marrow.—Determinations gave the following data: polymorphonuclear neutrophils (adult), 8 per cent; polymorphonuclear neutrophils (young), 14 per cent; metamyelocytes, 10 per cent; myelocytes, 8 per cent; myeloblasts, 3 per cent; primitive blasts, 26 per cent; lymphocytes, 1 per cent; eosinophils, 1 per cent; monocytes (adult), 3 per cent; monocytes (young and blast forms), 6 per cent; endothelial cells (phagocytic), 1 per cent; hemohistiocytes, 1 per cent; hemohistioblasts, 1 per cent; megaloblasts, 1 per cent; basophilic normoblasts, 10 per cent; eosinophilic normoblasts, 4 per cent; macropolycytes, 2 per cent. Megakaryocytes were present.

Fig. 6.—Photograph of the left kidney (*A*) and the right kidney (*B*), showing marked thickening of the capsule and invasion of the pelvis by neoplastic tissue.

The bone marrow was not grossly abnormal except for the presence of the cells of the monocyte group, 9 per cent of these being present, whereas normally usually less than 1 per cent is noted. The impression was that these cells had invaded the bone marrow, possibly from some external source, rather than that they were growing in the marrow. The immature monocyte can be differentiated in the bone marrow from the metamyelocyte, which it most resembles, by the fact that there is no sharp nuclear membrane in the former, and two or three nucleoli are prominent; the cytoplasm is spotty blue, with no granules or any element of pink such as is present in the metamyelocyte. The nucleus is round, occupies about one third of the area of the cell and in the films was usually eccentric.

Pathologic Diagnosis.—The diagnosis was: reticulocytoblastoma involving the retroperitoneal tissues, the kidneys, the spleen, the lymph nodes, the heart, the lungs, and the orbital tissues; neoplastic lipoid histiocytosis, and terminal lobar pneumonia.

REVIEW OF THE LITERATURE

In reviewing the literature relative to this report, chloroma, monocytic leukemia and the reticulo-endothelial system have been considered.

Fig. 7.—Photomicrograph of the neoplasm involving a mesenteric lymph node. The cells of the neoplasm are lying in connective tissue reticulum, giving the general appearance of a granuloma.

Chloroma is a rapidly growing neoplastic involvement of the hematopoietic tissues and is usually associated with a leukemic blood picture. It occurs most often in the young and is invariably fatal, with an average duration of from three to five months. The etiology is unknown. The

disease is characterized anatomically by tumors, which may or may not be green and which have a predilection for the skeletal system, especially the bones of the skull and more particularly those of the orbit. The clinical features are due to the tumors and the associated leukemia.

The literature of chloroma has been well reviewed by Brannan,[1] Barnert,[2] Washburn,[3] and Lehndorff.[4] The first case of chloroma was reported by Allen Burns, in 1821.[5] The fourth case was reported in 1853 by King, who gave the disease its name of chloroma. In 1885 von Recklinghausen suggested the relationship between chloroma and leukemia, and in 1904 Dock and Warthin[6] definitely established this relationship. In 1903 Türk published the first description of myeloid chloroma. Prior to this chloroma had been generally accepted as a disease of lymphoid tissue. In 1905 Sternberg stated the belief that chloroma is a primary disturbance of the lymphohematopoietic system and also of the blood-forming tissues. Subsequently the literature was reviewed and brought up to date by Lehndorff in 1910, by Boots in 1917,[7] by Brannan in 1926 and by Washburn in 1929. With the latter the number of cases totaled 162.[3] As studies of the blood became more accurate, cases of lymphogenous chloroma became fewer. Lehndorff[4] stated that since 1907 only myelogenous chloroma has been seen, and it is now believed that many of the cases described originally as instances of lymphatic chloroma were actually cases of myeloid chloroma. In only six of thirty-two cases reviewed by Washburn between 1926 and 1929 was the condition considered lymphogenous, and in only two of the six was the evidence conclusive.[3] Brennan[1] stated the belief that aleukemic phases in the disease were common.

Chloroma has been found to be more common in males than in females and to be especially frequent in the very young, though a few cases in persons in the fifth and sixth decades have been reported.[1] In sixty-eight of the seventy cases reported in Lehndorff's review the patients were 30 years of age or under, the average age being 18.8 years. The etiology is unknown, there being no evidence of heredity, as the

1. Brannan, Dorsey: Chloroma: The Recent Literature and a Case Report, Bull. Johns Hopkins Hosp. **38**:189-216 (March) 1926.

2. Barnert, Cyril: Report of a Case of Chloroma of the Orbit, Arch. Ophth. **53**:454-469 (Sept.) 1924.

3. Washburn, A. H.: Chloroma: Report of a Case with Recovery Following Roentgenotherapy, with a Review of the Literature, Am. J. Dis. Child. **39**:330-348 (Feb.) 1930.

4. von Lehndorff, H.: Chlorome, in Kraus, F., and Brugsch, T.: Spezielle Pathologie und Therapie innerer Krankheiten, Berlin, Urban & Schwarzenberg, 1920, vol. 8, pp. 572-626.

5. Burns, cited by von Lehndorff.[4]

6. Dock and Warthin, cited by Brannan.[1]

7. Boots, cited by Washburn.[3]

condition occurs chiefly in normal children [4] and trauma when present is usually coincidental in calling attention to a preexisting condition.[2] Syphilis, tuberculosis and nonspecific infections may be coexistent.[4] There is no clinical typical disease picture of chloroma.[8] Rather it is a symptom complex, the most typical sign being chloroma of the skull. In this condition there is a tendency to involvement of the skeletal system, especially of the head and face. In half of the cases the disease is of this class and occurs in children. Exophthalmos is probably the most common finding; it is due to the orbital tumors. It is usually progressive and is accompanied by swelling of the lids and conjunctival chemosis. Drying [4] of the cornea, with subsequent ulceration and opacity, may result from exposure due to extreme proptosis. Loss of motility varies from slight loss of motion in a single field to complete external ophthalmoplegia [1] and is usually due to orbital infiltration. Optic neuritis or papilledema may be present, followed by atrophy if the patient lives long enough. Visual acuity may be unaffected or may progressively diminish to ultimate blindness. Exudation into and about the nerve head occurs and is accompanied by hemorrhages, which may be under the hyaloid membrane, in the retina or on the disk. There are usually distention and tortuosity of the retinal veins. So far as the ocular findings are the result of the leukemia, Goldbach [9] reported, in addition to the aforementioned, such ocular findings as retinitis, sluggish reaction of the pupils to light, anisocoria, mydriasis, conjunctivitis, scleritis, blepharitis, glaucoma, iritis, keratitis, cataract, transitory exotropia and nystagmus. A second type of chloroma is that involving the ears, with dizziness, vertigo, diminution of hearing and facial paresis or paralysis. Diminished hearing was present in ten of twenty-two cases reviewed by Dock and Warthin.[10] During the course of the disease new tumors appear, especially in the bones of the skull, and when they occur under the temporal muscles they produce the "facies chloromatosa or frog's face." [4] Pearson [11] reported symptoms due to chloromatous infiltration of the temporal bones in twenty-five of forty-two cases. These were infections of the middle ear, tinnitus, hemorrhages, reduced hearing and pain in the mastoids. Washburn reported aural polypi with pain in his case.[3] Associated lateral sinus thrombosis and abscess of the brain were noted by Lehndorff.[4]

As a rule, the younger the patient the more rapid the course.[2] Leukemia, when present, is usually marked, with purpuric manifestations

8. Chloroma, in Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 3, pp. 2066-2068.

9. Goldbach, Leo J.: Leukemic Retinitis, Arch. Ophth. **10**:808-817, 1914.

10. Dock and Warthin, cited by Pearson.[11]

11. Pearson, W. W.: Chloroma with Special Reference to Ear, Nose and Throat Manifestations and a Report of Two Cases, Ann. Otol., Rhin. & Laryng. **39**:806-819, 1920.

in the skin and mucous membranes. There is a tendency to hemorrhage, which may be spontaneous or follow minor trauma, such as the extraction of a tooth. High grade secondary anemia develops, with associated asthenia, pallor and exhaustion. Clinically the liver, spleen and lymph nodes may be enlarged, and there may be tumors of any of the viscera as well as of the skin. There may be tenderness on pressure over the long bones, but there is no record of spontaneous fracture.[12] There is often a septic type of temperature.[13] Lesions of the mouth and pharynx are known, with necrosis and ulceration which may simulate diphtheria. Infiltrations of the tonsils and uvula occur.[4] Concomitantly there may be marked cervical lymphadenopathy.

The tumors may be well developed before the characteristic changes in the blood appear, and aleukemic phases of the disease have been noted.[1] Callender [14] noted that following irradiation therapy an aleukemic phase may be transformed into full-blown leukemia. This may also accompany an intercurrent infection or an operation.[4] The total white cell count is usually increased, with a tendency to predominance of young forms. The highest count reported, by Burgess,[15] was 1,880,000; and the lowest, by Pissavy and Richet, 3,200. There is a rapid decrease in the hemoglobin content and in the number of platelets.

Pathologically the tumors arise from the localized growth of leukemic deposits [1] or metastases, and the tumor cells metastasize in the blood to produce leukemia. Postmortem examination has shown widespread green growths, firm or hard, with a predilection for periosteum, the dura and the ligamentous structures, especially of the trunk. The short spongy bones are chiefly involved, and those of the skull and face are affected most frequently. Large flat, infiltrating masses are often present along the spine and in the sacral concavity.[1] Masses in the ribs and sternum are common. The bone marrow is usually hyperplastic, showing infiltration by the neoplasm. Its color may vary from gray-red with green patches to solid green. Direct continuity between the bone marrow and the periosteal tumors in the spine has been noted.[11] No organ or tissue is exempt, but the major sites of distribution in twenty-eight cases [2] were: head, twenty-six; periosteum of the orbit, thirteen; sinuses, nine; auditory area of the temporal bone, seven; temporal fossa, seven; ribs, eight; bone marrow, eight; liver, nine; kidney, thirteen; lymph nodes, twelve; spleen, four; intestine, four. Most organs show the characteristic infiltration of leukemia. The type of cell of the

12. Barnert.[2] von Lehndorff.[4]

13. Bamforth, J., and Edwards, J. L.: Chloroma: An Account of Four Cases, Lancet **1**:857-861 (April 22) 1933. Lehndorff.[4]

14. Callender, G. R.: Tumors and Tumor-Like Conditions of the Lymphocyte, the Myelocyte, the Erythrocyte and the Reticulum Cell, Am. J. Path. **10**:443-466 (July) 1934.

15. Burgess, cited by Brannan.[1]

tumors, the infiltrations and the blood [1] has been large undifferentiated mononuclears designated as myeloblasts or lymphocytes. Theories as to the origin of the green color are that it is a lipochrome or that it is hematogenous, but nothing is actually known of its chemical nature. Though some of the involved tissue usually shows the typical green color, Lehndorff expressed the belief that its presence is not absolutely necessary to make a diagnosis of chloroma.

The diagnosis is difficult unless definite superficial tumors exist.[16] Otherwise the findings might all well be ascribed to leukemia. The oxidase reaction serves as a means, though not an infallible one, of identification of the type cell. The granular cytoplasm of myelocyte stains bluish, giving a positive oxidase reaction.[17] The nongranular cytoplasm of the lymphocytes gives a negative reaction. Mature monocytes give a positive reaction, but the granules are finer and fewer and are usually grouped about the incisura of the nucleus. The young forms of the monocyte series usually give a negative oxidase reaction.[17] Supravital stains are invaluable in the differentiation of the living cells, as set forth by Sabin,[18] especially in difficult cases.

The diagnosis depends, then, on the careful study of blood smears and the histologic structure of the tumor and bone marrow. Differentiation between chloroma and myeloma is based on the usual blood picture of leukemia and the greenish color of the tumors in the former and the tendency to spontaneous fracture in the latter.[16] As a rule, the Bence-Jones type of protein is present in the urine of patients with myeloma and only rarely in that of patients with chloroma. Other tumors of bone, such as osteosarcoma, must be excluded. In cases in which the cervical glands are enlarged tuberculosis must be ruled out. Chloroma has been mistaken for lymphosarcoma and carcinoma of the breast. An eye has been enucleated in a case in which the condition has been chloroma.[4] Tumor of the brain may occasionally have to be ruled out.

The treatment of chloroma is largely symptomatic. Transfusions were of value in a case reported by Washburn [3] in which the disease was tentatively cured by irradiation. In this case small doses of the roentgen rays were given over a long period. There was no recurrence after a lapse of two and one-half years. The tumors are radiosensitive.[1] Solution of potassium arsenite has been used in the usual graded doses, without definite effect. Other arsenicals have been used, as have injec-

16. Chloroma, in Delafield, F., and Prudden, T. M.: Textbook of Pathology, ed. 14, Baltimore, William Wood & Company, 1927, p. 547.

17. Cohen, Martin: A Case of Chloroma with Ocular Lesions, Arch. Ophth. **57**:238-245 (May) 1928.

18. Sabin, Florence R.: Studies of Living Blood Cells, Bull. Johns Hopkins Hosp. **34**:277-288 (Sept.) 1923.

tions of mild mercurous chloride. Various oils and ointments have been used to keep the cornea from drying in cases in which there is marked exophthalmos.

Monocytic leukemia is at the present generally accepted as a clinical entity, though the origin of the monocyte remains controversial. Reschad and Schilling-Torgau [19] reported the first case of this type in 1913. Dameshek [20] in 1930, in a review, listed eighteen proved cases, and Clough [21] in 1932 gave a complete summary of twenty-three cases, with one of his own. Foord [21a] recorded the cases that occurred subsequent to 1933, bringing the total number of cases to thirty-three, in twenty-one of which postmortem examination had been carried out.

Clinically, monocytic leukemia differs only slightly from the other acute leukemias. It occurs most often in males,[18] appearing at any age from 5 to 78 years, in the cases reviewed by Dameshek, in most cases occurring between 30 and 50 years. Its etiology is wholly unknown, and the theory that it is due to infection, favored by fever, the rapid course and the almost invariable gingivitis, does not explain the mortality, the proliferative tendency and the fact that cultures of the blood have been negative in several cases.[20] The onset is usually gradual, and is marked by fatigue or some preceding infection, such as gingivitis or angina, but at times it is acute and fulminating, with a septic type of fever. The disease progresses rapidly after the initial symptoms appear. The gums become swollen and often ulcerate or become gangrenous. The fever becomes sustained. Pallor is marked, owing to marked anemia. Glandular involvement may be present, especially in the cervical region. The liver is usually slightly enlarged, and the spleen is almost always felt below the costal margin.[20] Purpura and bleeding [21] are common in the terminal stages and are often prominent signs throughout the course. The disease lasts between seven days [20] and six months,[20] ten weeks being the average duration. In two cases reviewed by Clough [21] the patient showed a remission after irradiation, and the course became more chronic. Farrar and Cameron [22] reported a case in which a brief remission followed transfusions, though the disease terminated fatally. The blood picture shows a white count of from 1,900 to 416,000, as noted

19. Reschad and Schilling-Torgau, cited by Dameshek.[20]

20. Dameshek, William: Acute Monocytic (Histiocytic) Leukemia: Review of Literature and Case Reports, Arch. Int. Med. **46**:718-740 (Oct.) 1930.

21. Clough, Paul W.: Monocytic Leukemia, Bull. Johns Hopkins Hosp. **51**: 148-177 (Sept.) 1932.

21a. Foord, Alvin G.; Parsons, Lawrence, and Butt, E. M.: Leukemic Reticulo-Endotheliosis (Monocytic Leukemia) with a Report of Cases, J. A. M. A. **101**: 1859 (Dec. 9) 1933.

22. Farrar, G. E., and Cameron, J. D.: Monocytic Leukemia with Data on the Individuality and Development of the Monocyte, Am. J. M. Sc. **184**:763-767 (Dec.) 1932.

by Sydenstricker and Phinizy.[23] The average white cell count is lower than in the other two types of leukemia, being from 15,000 to 45,000. Sudden marked changes in the count are more common, as are aleukemic phases.[20] The percentage of mononuclears exceeded 60 per cent at some period in all but two of the cases reported by Clough. There are usually some immature forms of the granulocytic series in the blood smears, due, no doubt, to the disturbance of bone marrow function. There are a uniformly progressive anemia and reduction in the number of platelets.[21]

The diagnosis is made almost wholly on the basis of the results of morphologic examination of the blood. Forkner [24] expressed the belief that the lesions of the mucous membranes are a differential point in the acute leukemias. In monocytic leukemia usually the patient shows a diffuse marked swelling of the mucosa, especially of the gingivae, with frequent cellulitis about the lesions, which extends often into the deeper tissues of the face. Because of the oral involvement these patients are usually seen first by a dentist. In eighteen of twenty-two cases reported in Clough's review the patients showed marked stomatitis or pharyngitis,[24] with swelling of the gums and, in the majority of cases, ulceration. Alveolar and sinusal abscesses were present. Oral and pharyngeal involvement occurs, in chloroma, but with less frequency than in monocytic leukemia. The size of the spleen in monocytic leukemia, according to Forkner, is midway between that of the usually greatly enlarged spleen seen in acute myeloid leukemia and that of the slightly enlarged spleen seen in acute lymphatic leukemia.[25] The lymph glands are generally enlarged in lymphatic leukemia; they show a tendency to localized enlargement, as in the cervical region, in monocytic leukemia. The type cell in monocytic leukemia [21] is seldom a mature monocyte but usually an immediate precursor, including the most immature cell or histiocyte. These cells are larger than any normal blood cell; in moist preparation they show marked motility,[18] and on supravital staining, active ingestion of dye. Pseudopodia and scalloping of the edges testify to the motility.[21] The cytoplasm is blue-gray and has the appearance of ground glass, with typical monocytic granules first seen about the nucleus in the youngest cells. The nucleus is large, oval, indented, bean shaped or even folded so as to appear almost lobed. The chromatin is in a fine spongelike meshwork, which becomes coarse and skeinlike in the more mature cells.

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23. Sydenstricker, V. P., and Phinizy, T. B.: Acute Monocytic Leukemia: Case with Partial Autopsy, Am. J. M. Sc. **184**:770-777 (Dec.) 1932.

24. Forkner, Claude E.: Clinical and Pathologic Differentiation of the Acute Leukemias, with Especial Reference to Acute Monocytic Leukemia, Arch. Int. Med. **53**:1-34 (Jan.) 1934.

25. McCrae, T.: Leukemia, in Osler, W., and McCrae, T.: Principles and Practice of Medicine, ed. 10, New York, D. Appleton and Company, 1927, p. 756.

Phagocytosis with red cells and cellular fragments has been reported.[18] The immature cells, as a rule, give a negative oxidase reaction, and the mature cells, a slightly positive oxidase reaction.[20]

The differential diagnosis should include the other two acute leukemias, infectious mononucleosis and agranulocytosis. The identification of the type cell establishes the form of leukemia. Infectious mononucleosis is ruled out by the appearance of a large number of histiocytes in the blood. There is rarely diminution of the cells of the granulocytic series so marked as in agranulocytosis.

Autopsy shows [21] extensive leukemic infiltration of organs, especially of the lymph nodes and spleen, with cells similar in type to monocytes of the blood, which are regarded as reticulum cells. There are necrotizing [21] lesions of the mouth and throat. The liver and spleen are usually enlarged, and the bone marrow is hyperplastic. There are evidences of hemorrhages in the skin and serous surfaces. The kidneys are swollen, owing to growth of the reticulum cells. Frequently the mesenteric lymph nodes show extensive involvement. The striking features are the widespread distribution of the process and the tendency in many organs for the process to center in the loose adventitial and periadventitial tissues.[21] In the hili of some lymph glands Clough reported an invasive type of growth somewhat resembling a malignant neoplasm. There is a widespread distribution of mitosis, indicating active cellular proliferation and not passive accumulation from the circulating blood. One report [21] of a typical case in which autopsy was limited to an abdominal incision described mononuclear infiltration of the type cell in the spleen, mesenteric lymph nodes, bone marrow, liver, gallbladder, kidney, prostate, bladder, pancreas, intestine, lungs, heart, aorta and muscle.

Piney [26] included in the reticulo-endothelial system many of the cells of the splenic pulp, medullary follicles and cords, of the lymphatic glands and other lymphatic structures, of the splenic sinuses, of the liver capillaries (Kupffer stellate cells), of capillaries in the formative bone marrow and of the adrenal and pituitary glands. Particles of dye are observed in the endothelial cells and also in elongated reticular cells which in these locations are associated with them. These two types of elements are included under reticulo-endothelium. Normally these cells are considered to have a destructive function of importance in keeping the number of red cells constant. They are also concerned with the formation of bile and the storage of fatty materials and are believed by some to participate in the processes of immunity. Pathologic processes involving this system are Hodgkin's disease, a nonleukotic proliferation; monocytic leukemia, a leukotic proliferation, and Gaucher's and Nie-

26. Piney, A.: Recent Advances in Haematology, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1928.

mann-Pick's disease, a proliferation due to storage. Piney described a pathologic overgrowth of this system of unknown origin in which there was an associated leukemic blood picture of large monocytes. Aleukemic proliferations also occur. He expressed the belief that the existence of true blastomatous neoplasms of the reticulo-endothelial system is determined only if a morphologic feature is present for recognition, which must be taken to be the formation of fibrils. These fibrils emerge from the endothelial cells and form a fine network in the intercellular spaces. Those who believe the monocyte to be derived directly from the reticulo-endothelial system will include monocytic leukemia as a proliferation of the reticulum cells. Callender [14] favored this view and added that the tissues in such cases show a proliferation characterized by a delicate meshwork of reticulum closely surrounding the individual cells. Goldzieher and Hornick [27] reported a case of reticular hyperplasia "of the third main constituent of the hemato-poietic tissues," which may be leukemic or aleukemic, with the type cell the monocyte.

Caldwell [28] suggested that types of cells which react actively in hyperplasia and which have such diverse possibilities for functional differentiation as characterizes the cells of the reticulo-endothelial system would be expected to serve as a fertile source of neoplasms. Some of these, he stated, are of frequent occurrence, and their origin from the reticulo-endothelial system [28] has been recognized for a long time. An illustration is the reticulum cell sarcomas which take their origin from the reticulum cells of the primordial follicles of the lymph glands and possibly also from the sinus reticulum. Callender [14] described the reticulum cell sarcoma as aleukemic, with mononuclears increasing at times after roentgen therapy, without enlarged nodules and without true metastases. Microscopically there is diffuse hyperplasia of the reticulum cells, obliterating the structure of lymph nodes in varying degree, and a fine meshwork of reticulum about the individual cells.

SUMMARY

The conjecture as to whether the monocyte originates from the reticulo-endothelial system is not within the scope of this paper to consider.

In the case reported the patient, a man 55 years of age, presented a leukemic blood picture, with the type cell the monocyte, in association with a neoplastic process invading both orbits. The onset was gradual, fatigue on use of the eyes being the first symptom noted. Subsequently

27. Goldzieher, M. A., and Hornick, O. S.: Reticulosis, Arch. Path. **12**:773-782 (Sept.) 1931.

28. Caldwell, George T.: The Reticulo-Endothelial System and Its Relation to Neoplastic States, South. M. J. **27**:205 (March) 1934.

there were exophthalmos, gingivitis, diminution of vision and hearing, petechial hemorrhages and a septic temperature. The total white cell count varied between 12,350 and 26,400. The percentage of monocytes varied from 20 to 62. The lowest hemoglobin content and red count recorded were 61 per cent and 3,760,000, respectively. The course was of approximately five months' duration. The condition terminated fatally. It may have been somewhat prolonged by the irradiation therapy.

Biopsy of orbital tissues showed a neoplastic infiltration, which was diagnosed as reticulocytoblastoma or some form of chloroma or myeloma. Autopsy showed a diffuse neoplastic infiltration the distribution of which conformed to that of chloroma. The sections of tissue showed the cells of the neoplasm in a connective tissue reticulum, satisfying the criteria for true blastomatous neoplasms of the reticulo-endothelial system as set forth by Piney. The pathologic diagnosis was reticulocytoblastoma. Centrally some of the mesenteric nodes showed the green color characteristic of chloroma. The process involved the orbital and retroperitoneal tissues, kidneys, spleen, lymph nodes and heart. There was in addition a neoplastic lipoid histiocytosis, involving especially the retroperitoneal tissues.

For the disease entity presented by this patient we suggest the name monocytic chloroma.

DEPOSITS OF FAT IN TRACHOMATOUS PANNUS

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Among the various changes of the cornea in trachoma united under the general name of pannus or interpreted as sequels of it there is one form of damage of the cornea which is of great interest. It is characterized by the appearance of pale yellowish spots which are situated mainly in the center of the cornea.

Among all the authors of textbooks and manuals, only Fuchs [1] has mentioned these lesions. He wrote about changes in pannus of long standing in the form of "small, intensely white spots in the region of the pupil." The appearance of those spots, which "closely adhere to the thin vessels of the pannus, resemble leaden incrustations. The spots lie superficially and can be removed by scraping."

These changes were described in detail by Calderaro [2] from the Corinesno clinics. According to Calderaro, these lesions are rather rare and were observed in only 0.1 per cent of all cases of chronic trachoma. The lesion begins at the center of the cornea, nearer to the lower border of the pupil than to the upper, in the form of small spots, like strokes of a fine paint-brush. They are situated beneath Bowman's membrane (around the ends of the vessels of the pannus). Next to the spots which appear first, new ones appear, spread in various directions, enlarge and come into contact but do not become fully confluent. The spots not only spread on the surface but penetrate into the deeper layers of the cornea, as though pushing each other away.

When in the progress of the disease the opalescence reaches the paracentral portion of the cornea, the spots assume a rather characteristic appearance. The more transparent the periphery of the cornea is, the more conspicuous the milky color of the spots becomes. Older lesions appear more yellowish and have a silky luster and irregular outlines. The curvature of the cornea is usually not affected. In some cases, however, especially over the yellowish spots, an elevation of the epithelium may be observed. The blood vessels of the pannus, subdividing in branches, frequently form a dense network, the smallest

1. Fuchs, E.: Lehrbuch der Augenheilkunde, ed. 12, Leipzig, F. Deuticke, 1910, p. 164.

2. Calderaro, S.: Arch. f. Augenh. **69**:35, 1911.

branches of which are bordered by grayish streaks, which gradually taper and disappear. Even in cases in which the condition is of long standing and progressive the lesions do not extend over the entire cornea but remain limited to the central and the paracentral portion.

For diagnostic purposes Calderaro performed partial trepanation of the cornea in ten cases. For fixation he used mainly Müller's fluid, followed by washing with a 60 per cent solution of alcohol for forty-eight hours; for staining of the fat he used osmic acid. He found the fat mixed with hyaline granules. Fat was present only in old spots, which explains the yellowish appearance of the older spots.

In two cases published by Takayasu [3] changes were present similar to those described by Calderaro. In both cases whitish round or oval spots were seen, the older ones having a yellowish tint. Slit lamp examination showed that the spots consisted of fine fibers with serrate edges. Most of the spots were superficial, and with the corneal microscope one could see a clear area between the spots, the deeper layers of the cornea being transparent. The blood vessels arose from the limbus without branching, and only in the central area did they divide into smaller branches above or between the spots.

On histologic examination Takayasu observed the corneal lamellae in some places in a state of breaking down. The débris took the specific sudan III stain. Both the patients were girls, of 14 and 16 years, respectively, who had suffered from severe trachoma since childhood. In spite of this fact Takayasu considered his cases as instances of primary fatty degeneration on the basis of malnutrition and denied that trachoma was an etiologic factor. Similar cases were not observed in Japan, where trachoma is common. Nevertheless, on the basis of the description of the lesions, one should consider Takayasu's cases analogous with those of Calderaro. To the cases of fatty deposits may be added three cases described by Balod [4] from Rubert's Hospital. From the point of view of the clinical aspects these cases coincide with those of Calderaro, but on histologic examination Balod observed hyaline degeneration. The hyalin was distributed in large lumps or small granules around the blood vessels, and deposits of calcium were seen. Balod did not report that he observed fat, but he explained the changes described by him as manifestations of the tendency of the trachomatous process to hyaline formation. This tendency was emphasized in the description of three cases of hyaline degeneration reported by Rubert [5] and others.

3. Takayasu: Arch. Ophth. **82**:475, 1912.
4. Balod, K.: Klin. Monatsbl. f. Augenh. **78**:362, 1927.
5. Rubert, J.: Arch. f. Augenh. **65**:271, 1910.

Apparently Capolongo[6] had in mind the same changes when he reported two cases of cicatricial trachoma with central clouding of the cornea, which had the appearance of deposits of calcium but in reality consisted of cellular deposits.

In the period of 1927 to 1929 I examined about five hundred patients with pannus with the slit lamp. In sixteen cases I observed changes as described by Calderaro. I shall not encumber my article by the description of all these cases. I consider it necessary, however, to report the most characteristic ones.

REPORT OF CASES

Case 1.—M. M., 19 years old, had been suffering from trachoma for several years. On Nov. 19, 1928, there were fresh scars on the outer canthus of each eye, following canthoplasty. The palpebral conjunctiva was smooth and showed some scars. The conjunctiva of the lower lids was wrinkled. The right upper lid showed irregular growth of eyelashes and a narrow strip of transplanted mucosa from the lip. The operation was the procedure of Millingen and Canencko. In the center of the cornea of the right eye was a yellowish white opacity in the shape of small plaques situated in the superficial layers along two large blood vessels. A more pronounced opacity was present in the left eye.

Neither eye showed any changes in the iris. The pupils were equal and narrow, and the fundus could not be seen on account of the opacity of the cornea. Vision was 6/200 in the right eye and 9/200 in the left.

On December 6 partial transplantation of the cornea of the right eye was performed according to the method of Duverger and Velter.[7] An oblong strip of cornea was cut out with a lancet, somewhat below and externally to the center, and in its place a flap was transplanted that was taken from an eye which had been removed on account of a choroidal tumor. The flap was well fitted. Its lower external end was held in place by a thin suture. During the manipulations Descemet's membrane perforated, and the aqueous escaped.

On December 7 there was marked blepharospasm. On December 9 the flap was in place. On December 12 the eye was congested, and the pupil was narrow. On December 19 the congestion was subsiding; the pupil dilated poorly. On December 28 the vessels began to grow into the graft. On Jan. 6, 1929, the congestion was negligible; the corneal graft was cloudy and vascularized.

On January 9 an operation for trichiasis was performed on both upper lids, with transplantation of mucous membrane of the lip. On January 16 vision was 7/200 in the right eye and 4/35 in the left, and the patient was discharged.

On February 18 the patient was readmitted. The scleral vessels were dilated; the graft, which was opalescent at the periphery and clear at the center, healed beautifully. The pupil was regular in form.

On February 20 the Pirquet reaction was strongly positive. On March 2 vision was 4/200 in the right eye and 8/200 in the left, and on June 2, 1931, vision was 20/100 in the right eye and 20/200 in the left.

Brilliant dots were seen in various layers of the cornea of the right eye, especially on the surface. Where Descemet's membrane had been perforated a tree-like branching was seen.

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6. Capolongo: Klin. Monatsbl. f. Augenh. **46**:200, 1911.

7. Duverger and Velter: Therapeutique chirurgicale ophtalmologique, Paris, Masson & Cie, 1926, p. 247.

On Nov. 15, 1934, the upper third of the cornea of the right eye was opaque and covered with blood vessels; there were also blood vessels below, going from the limbus toward the graft. The cornea was opaque superficially, the central portion being the clearest, without any vascularization. No whitish spots were noted, but in the lower part of the cornea, on the border of the transplanted flap, minute dotlike whitish deposits were seen. They were situated in the middle and deep layers. At the same place were folds of Descemet's membrane, spreading like a fan from the point of perforation. In the other parts the outlines of the transplanted flap were not noticeable.

The superficial opacity of the cornea of the left eye was more pronounced, the central portion being more opaque than the periphery. In this eye only one small blood vessel reached the center of the cornea.

On May 25, 1935, vision was 0.3 in the right eye and 0.4 in the left. On examination with the slit lamp no whitish deposits could be observed in either eye. No blood vessels were noticed. There was a slight superficial opacity in both corneas.

Case 2.—T. V., 21 years old, was admitted to the hospital on Feb. 21, 1929. Vision was 9/200 in the right eye and 8/200 in the left. There was a history of long-standing disease of the eyes. Three years previously the patient had been treated in the eye clinic of Gomel for four months. The right eye was operated on. Vision became worse, especially during the month previous to admission. The patient's mother had trachoma and trichiasis.

Both eyes showed slight blepharophimosis; a small amount of pus was expressed from the lower right lacrimal points. The conjunctiva of the lids showed scars and was rather smooth. The tarsus was thickened and distorted. The fornices were shortened. Trichiasis and distichiasis of the upper lids were present, due to entropion. The scleral vessels were dilated. The corneas were opaque and vascularized by superficial large and small vessels. The anterior chamber was of normal depth; the pupils were narrow and reacted to light. The iris did not show any changes. The fundus could not be seen.

On the cornea of the right eye below the border of the pupil there was a yellowish white opacity in the form of separate and confluent plaques which were situated along the branches of a large vessel and also along the smaller ones. These spots had an archlike arrangement and surrounded the paracentral part of the cornea.

On the cornea of the left eye below the pupillary area there was an opacity in the form of separate small plaques, and some dotlike opacities were present in the pupillary area. Such opacities were also situated along the branches of the blood vessels which came from the limbus.

The Pirquet reaction was strongly positive.

Treatment consisted in massage with yellow ointment (yellow mercuric oxide ointment) and probing of the ducts.

On June 8 vision was 4/25 in the right eye and 4/20 in the left.

Nothing could be expressed from the lacrimal points of the right eye. The lacrimal canal was patent on syringing.

On November 29 there were widely branching blood vessels in the periphery of the cornea of the right eye, which originated from three conjunctival vessels, at 12, 3 and 7 o'clock. The blood vessels were wide, full and very tortuous and formed numerous anastomoses. The cornea was diffusely opaque in the periphery. The whitish deposits had spread upward and now occupied the entire central portion of the cornea, forming a round plaque, which became more intensely white in the lower part. These opacities showed transparent streaks, which corresponded to

the continuation of the smaller vessels. In some places the vessels approaching the white masses were markedly changed. The blood column within them was narrowed; the walls became thicker and more transparent, and the small branches of the blood vessels gradually changed into the aforementioned light streaks. Both the vessels and the whitish masses were mostly situated in the superficial layers; some were in the middle layers. The most opaque deposits were in the lower part of the cornea. They were elevated above the surface of the cornea; in the remaning portions the cornea was smooth and even. The corneal microscope showed the cornea somewhat thinned in the center.

On June 9 the right eye was operated on. With Hippel's trephine a small section which was to be removed was marked almost in the center of the cornea; then the marked area was cut out with a lancet. In the place of the defect a small flap of cornea was transplanted from an enucleated eye; then one suture was applied.

On June 11 the flap was in good position, and the pupil was narrow. On June 14 the anterior chamber was normal, and the pupil was narrow. On June 19 the suture was removed. The pupil was dilated; the flap was transparent and well adherent; no blood vessels were seen. The anterior chamber was normal. On June 23 vision was 4/15 in the right eye and 4/10 in the left.

On superficial examination in May 1935 vision was 0.1 in the right eye and 0.4 in the left. The borders of the transplanted flap were not visible. Very small numbers of whitish deposits were noted on the lower border of the flap. The center of the right cornea was cloudier than that of the left. This explains the lower visual acuity of the right eye.

CASE 3.—I. L., 45 years old, was admitted to the hospital on May 19, 1929. Vision was 0.9 in the right eye, and light projection was correct in the left.

The palpebral conjunctiva of the left eye showed scars. The tarsus was thickened, and there was a large amount of mucus in the conjunctival sac. The cornea was vascularized by blood vessels which came from the limbus and showed extensive ramifications near the center of the pupillary area. There was an opacity, consisting of separate small plaques. The changes were of the same kind as those in case 1 and were located in the superficial and middle layers of the cornea. The anterior chamber was deep; the pupil was not visible.

The right eye showed evidence of cicatricial trachoma. The cornea was clear.

On June 9 the left eye was operated on. With Hippel's trephine a portion of the cornea was marked and cut out with a lancet. The defect was filled with a piece of cornea taken from an enucleated eye.

On June 10 the corneal flap was well in place. On June 12 the eye was considerably irritated; the pupil was not dilated. On June 16 congestion was still present, and there was a large amount of mucous discharge in the conjunctival sac. Superficial and deep blood vessels penetrated the edge of the transplanted flap. On June 26 vision was 0.9 in the right eye and 4/200 in the left.

CASE 4.—A. L. had a history of trachoma of many years' duration. Examination showed cicatricial trachoma of the conjunctiva.

The right eye showed negligible conjunctival congestion; numerous blood vessels spread from the entire limbus into the cornea and showed extensive ramifications and anastomoses in the center, forming a network of small vessels. The entire cornea was diffusely opaque in its superficial layers. In the central portion the opacity was more intense and consisted of pale yellowish islands, which were partly confluent. Around this central opacity were single whitish dots, situated along the smaller blood vessels. In some places, more so in the superficial corneal layers, the small vessels passing between these deposits were obliterated and appeared as narrow, transparent stripes within the opacity.

The changes in the left eye were of the same nature as those in the right eye, but the epithelium formed elevations so that the surface of the cornea appeared uneven.

On June 6 the right eye was operated on. With Hippel's trephine an area of the cornea was marked and cut out with a knife. The defect was filled with a corneal flap from an enucleated eye. The flap was held in place by a suture from the upper to the lower limbus.

On June 8 the flap was well in place. After the removal of the suture a linear opacity remained. On June 19 blood vessels were seen extending from the limbus toward an opaque ring which surrounded the transplanted flap.

I shall cite two other cases illustrated by drawings of the slit lamp picture (figs. 1 and 2).

CASE 5.—On examination the entire periphery of the cornea of the left eye of D. was seen to be covered with blood vessels, which formed a dense network

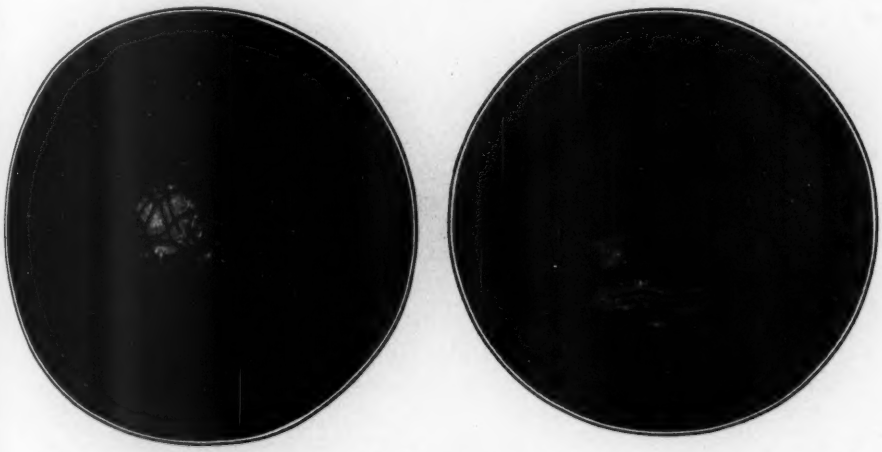

Fig. 1 (case 5).—Appearance of the corneas. *A* shows the cornea of the left eye, and *B*, that of the right eye.

in the center. The largest vessels were above and below in the nasal portion. In the periphery the blood vessels were superficial; they became deeper toward the center. The cornea was diffusely opaque. Toward the center of the cornea the opacity was more intense and became yellowish white. The borders of this opacity were irregular. Numerous blood vessels reached the central opacity. Some of them were obliterated and looked like transparent streaks dividing the opacity into small islands. Both in the central part of the cornea and in the periphery the blood vessels were bordered by small whitish deposits.

The sixth case is of interest because it is one in which there was a partial opacity.

CASE 6.—Y. A., 20 years old, was examined Aug. 3, 1929. The cornea was diffusely opaque, more so in the center than in the periphery. Three large white opacities were seen, one of which was less intense than the others. Several smaller opacities were present. The larger opacities were penetrated by blood vessels; the

smaller ones were situated in close vicinity to blood vessels. The entire periphery of the cornea was vascularized, the vessels forming a capillary network in the corneal center. When I saw this patient last (in 1934) I could not observe any of the white spots.

COMMENT

As I have already mentioned, of the five hundred patients with pannus whom I examined with the slit lamp, sixteen showed the changes described—obviously, not a small percentage.

Fig. 2 (case 6).—Appearance of the cornea.

The distribution of the cases according to the age of the patient is shown in the table. This enumeration shows that in 42 per cent of the cases the condition occurred at a comparatively early age.

Distribution of Cases According to Age

Age of Patient	Number of Cases
Up to 10 years	1
11 to 20 years	5
21 to 30 years	2
31 to 40 years	1
41 to 50 years	4
51 to 60 years	3

The picture of the condition is typical and is characterized by the following signs: The affected part of the cornea is usually the center. The opacities as a rule appear first below the pupillary area and spread upward. The healing process takes place in the opposite direction. The opaque spots in the lower part of the pupillary area remain longest. In the beginning the deposits consist of white spots with rather distinct outlines. They are situated along the blood vessels, mostly at the bifurcations. In their development the granules enlarge, come in contact and flow together, forming irregular plaques. As they grow they assume a yellowish tinge. The deposits are situated superficially; the larger

ones stand out somewhat above the surface of the cornea and frequently spread into the middle layers. In addition, peculiar changes of the blood vessels are noted. The vessels are congested, tortuous and slightly bluish; usually only two or three of the numerous vessels of the pannus show these features. Toward the center of the cornea the vessels divide freely and form a dense capillary network in the region of the deposits. The deposits form a kind of sheath around the walls of the finer blood vessels. At the same time the lumen of the blood vessels becomes narrower and is obliterated eventually, while the walls of the blood vessels become transparent. Consequently, transparent streaks are

Fig. 3.—Photomicrograph showing histologic changes due to pannus.

formed which cross the deposits like a network of canals. These changes may disappear completely. The process of absorption of the deposits may take a long time. The white spots and a number of the blood vessels usually disappear at the same time. The restoration of vision depends on the original site of the deposits and the extent of their resorption.

Figures 3 and 4 show histologic changes due to pannus. The epithelium is thickened, and its superficial layers are partly sloughed off. The outlines between the cells are indistinct. The basal cells appear elongated. In places where there is a superficial scar the epithelial layer is thin, and the cells seem pushed toward the side of the scar. Where the infiltration of the superficial layers is more pronounced nuclei of

leukocytes or parts of them are seen wandering through the epithelial layer. Bowman's membrane is intact in some places; in others it seems to be split into numerous lamellae which form shreds behind the epithelium. Immediately behind Bowman's membrane or behind the epithelium there are groups of cells—either round cells, if the process is fresh, or fibrobasts, if the process is an older one. In this layer there are also seen longitudinal sections of small capillaries, which are recognized by the endothelial cells. The corneal lamellae at the area of cicatrization are thin and irregular. The fat is located in the superficial and deeper layers. Deposits of fat were especially prominent in case 2. On staining with the sudan stain the fat shows the characteristic orange-

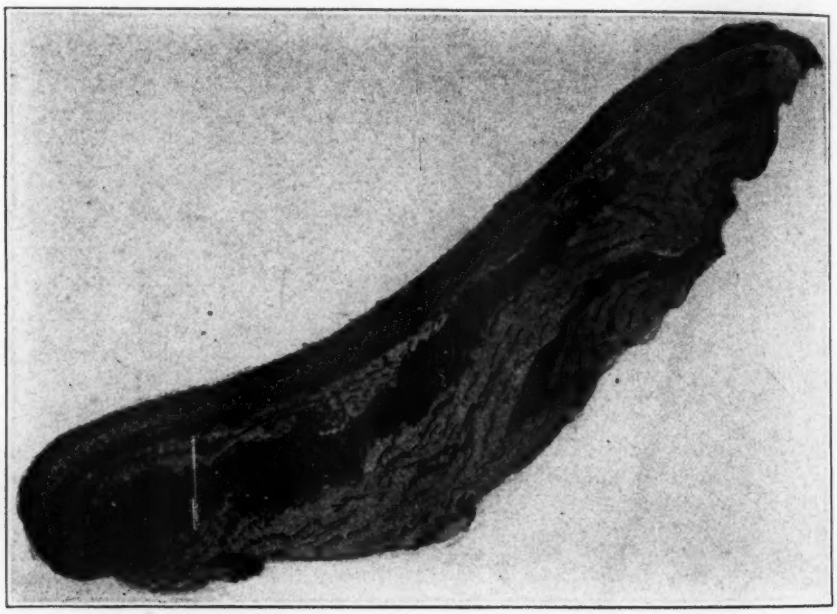

Fig. 4.—Drawing showing histologic changes due to pannus.

red color. It is seen as small granules in the protoplasm of the corneal cells and as larger granules between the lamellae. In some places the masses of fat become confluent and form large lumps, thus separating the corneal lamellae. Between the fat deposits are single cells which stain only with hematoxylin. In other cells the nucleus takes the hematoxylin stain, while the protoplasm stains with the sudan stain. As the amount of fat increases the cells become larger and are entirely stained with the sudan stain, the nucleus becoming invisible.

The fissures are surrounded by separate epithelial cells which have a more or less cubic form. One also observes cross-sections of vessels which are surrounded by large cuboid cells similar to those described

by Aschoff.[7a] According to Aschoff, these cells surround the walls of the blood vessels and give them the appearance of glandular tubules. The aforementioned fissures taper toward both ends and merge with the walls of the blood vessels. There is reason to assume that the formation of the fat takes place at the expense of the endothelial cells.

The opinions about the process of fatty degeneration of body tissues have undergone considerable changes in recent years. Virchow denied the possibility of fat formation in pathologic processes by way of infiltration and claimed that pathologic deposits of fat in the tissues have two sources: 1. Lipoid substances (fatty acids, soaps) are deposited together with proteins in the cell protoplasm; they are freed when the cell protoplasm degenerates. 2. The main source of fat formation takes place through the process of metamorphosis, in which the proteins and carbohydrates are transformed into fats.

Aschoff [8] denied the importance of these two forms of fat formation. According to this author, one is dealing only with the process of infiltration, both in physiologic and in pathologic conditions. The appearance of fat in places where it is not supposed to be or the increased amount of it where it is found ordinarily in smaller amounts is a process of accumulation of fat. Aschoff stated the belief that "fatty degeneration" in the sense of previous authors (Lukianov and others) does not exist, that the deposits of fat signify not a local process but a part of a generalized process and that the borderline between the primary and the secondary fatty changes (of the cornea) is not clearly defined. As to the presence of fat in the cornea, Aschoff expressed the belief that one is dealing with infiltration by fat.

Dreyfus [9] and Axenfeld [10] agreed with the theories of Aschoff. Axenfeld compared the intima of the blood vessels and the cornea. The hyaline deposits in the superficial layers of the intima resemble the nodular keratitis; the same resemblance exists between fatty deposits in arteriosclerosis (lipoidosis) on one hand and dystrophia adiposa corneae and arcus lipoides corneae on the other hand. In comparison with other organs of the body the cornea has the peculiarity of becoming impregnated with fat or calcium at an early age. It is possible that light rays have a certain influence. Axenfeld [11] described a case in which there were deposits of calcium in the cornea which formed disseminated spots over the entire surface and disappeared after the influence of light was eliminated.

7a. Aschoff, L.: Pathologische Anatomie, Jena, Gustav Fischer, 1911.
8. Aschoff, L.: Vorträge über Pathologie, 1925, p. 42.
9. Dreyfus: Ann. d'ocul. **170**:716, 1933.
10. Axenfeld, T.: Klin. Monatsbl. f. Augenh. **58**:58, 1917.
11. Axenfeld, T.: Klin. Monatsbl. f. Augenh. **85**:493, 1930.

As far as fat deposits in the cornea are concerned, Versé's[12] experiments on the artificial formation of arcus lipoides corneae are of great interest. Experiments on rabbits showed that deposits of neutral fats in the cornea occurred only in cases in which lipoids were given by artificial feeding together with cholesterol. There are certain kinds of tissues which absorb fat only in this combination with cholesterol, e. g., the tissues of the aorta and of the cornea. Such tissues are called cholesterolophilic. If a rabbit is fed butter without cholesterol, fat will be deposited in the muscles and in the heart but not in the cornea.

Versé's experiments were repeated and continued by Rohrschneider,[13] Kolen[14] and Vannas.[15] According to these authors, fatty changes of the cornea are of two types, peripheral and central. The peripheral type appears as the primary change and causes the formation of blood vessels. The deposits of fat in the center of the cornea are considered a secondary phenomenon.

There are other factors which are of importance, such as predisposition (Szily,[16] Kusama[17]), glandular disturbances (Denti[18]) and tuberculosis (Pirquet's reaction was positive in two of my cases).

Returning to a consideration of the cases which I have described, I shall point out the causes which facilitate the accumulation of fat in the cornea. A main factor is the presence of numerous blood vessels in the pannus. The theory of the importance of blood vessels in accumulation of fat is supported by Ricker.[19] According to this author, it is impossible to draw a line between a physiologic and a pathologic metabolic process. The latter appears as a continuation of the first. Formerly there was a distinction between simple infiltration by fat and fatty degeneration, the implication being that in cases of simple infiltration the cells were not injured, at least not in the beginning of the process, and that in cases of fatty degeneration the fat accumulated in degenerated, injured cells. Ricker denied the existence of such a distinction. He stated that he regarded as a common cause of any increased deposits of fat different degrees and stages of circulation of the blood. Normally fat tissue is distinguished from other tissues by the abundance of large capillaries and a slower circulation of the blood. Furthermore, fat can penetrate from the blood into the tissue fluids

12. Versé, M.: Klin. Monatsbl. f. Augenh. **74**:110, 1925.
13. Rohrschneider: Klin. Monatsbl. f. Augenh. **74**:93, 1925.
14. Kolen, A.: Klin. Monatsbl. f. Augenh. **86**:204, 1931.
15. Vannas, M.: Klin. Monatsbl. f. Augenh. **87**:289, 1931.
16. von Szily, A.: Klin. Monatsbl. f. Augenh. **71**:30, 1923.
17. Kusama, K.: Klin. Monatsbl. f. Augenh. **66**:111, 1921.
18. Denti: Ann. d'ocul. **167**:327, 1930.
19. Ricker, G.: Pathologie als Naturwissenschaft, Relationspathologie, Berlin, Julius Springer, 1924, p. 168.

only when it is broken up into its constituents. Resynthesis takes place in the cell protoplasm itself. In accordance with his general standpoint about the nervous regulation of the blood supply, Ricker came to the conclusion that the building up or the disintegrating of fat depends on the character of the blood flow, which is regulated by the nervous system. Thus two factors are influencing the appearance of fat in the tissues: (1) an increase in the amount of fat in the fluid penetrating into the tissues and (2) retardation of the circulation of the blood. According to Rohrschneider, both conditions are present at the time when arcus senilis appears in the cornea. In such cases an increase of fat in the blood was found. There is also a retardation of the blood stream due to the widening of the sum total of the capillaries and interlamellar lymph fissures of the cornea. The retardation is more sudden at the limbus than in the center of the cornea. Besides the local causes there are external conditions which may influence the appearance of fat. In commenting on his experiments on rabbits Rohrschneider pointed out that arcus senilis does not appear necessarily in both eyes at the same time but may occur earlier in one eye, depending on the size of the fissure of the lids and the pressure of the lid on the eyeball. According to Versé, local causes, such as mechanical, chemical or toxic injuries of the cornea, may be followed by the increase of deposits of fat. Thus, he obtained an increase of arcus lipoides corneae in cases of artificial injury to the eye by opening the anterior chamber with a lancet, by puncturing with the discission needle, by the application of silver nitrate and by provoking an infectious keratitis after the injection of cultures of Staphylococcus.

In my cases of deposits of fat in the cornea associated with pannus the conditions were present which are required by Ricker and his school. Unfortunately, I had no opportunity to examine the fat content of the blood of my patients, but the importance of these tests is limited. According to Engelking,[20] it is not always possible to prove the presence of fat in the blood even if it is present in pathologically large quantities. I pointed out the hyperemic stage of the pannus. The vessels were tortuous and dilated, and their walls were tense, thickened and highly light reflecting. This condition of an increased blood supply and retardation facilitates the appearance of fat in the cornea in cases of trachomatous pannus. Similar processes are observed in other diseases of the cornea. There are cases of interstitial keratitis in which the picture is similar to that in the cases already described, with the difference that the formation of whitish spots takes place in the deeper layers of the cornea. Under the slit lamp these spots appear broken up into small granules which are situated along the blood vessels, especially at their

20. Engelking, E.: Klin. Monatsbl. f. Augenh. **79**:721, 1927.

bifurcations; they partially surround several vessels with a whitish membrane and form a dense network in the posterior layers of the cornea. The observation of fat by Nakaizumi [21] on histologic examination of the cornea in cases of interstitial keratitis is analogous with the finding of deposits of fat in cases of pannus. In a case of prolonged keratitis of a different origin connected with considerable vascularization I have had the occasion to observe a similar picture.

In so-called keratitis disciformis (Fuchs), a disease which is not clearly defined, vascularization of the cornea from the limbus is present, which in a prolonged course spreads fanlike toward the center of the cornea. In the periphery of the corneal opacity whitish spots may appear which are characteristic fatty changes.

In the pathologic picture of deposits of fat the cause and the end-results are of great interest. Calderaro, Takayasu and others came to the conclusion that regression does not occur in the process of infiltration by fat. The cases described by me demonstrate clearly the contrary and prove clinically the theories of Ricker, Aschoff, Axenfeld and others. In all my cases which were under prolonged observation (for two or three years or longer) I observed disappearance of the spots with simultaneous clarification of the cornea and improvement of vision. In cases 1 and 2 vision improved from 0.05 to 0.3 and 0.4, respectively. Vision in these two cases improved independently of the operation. The cornea that was not operated on cleared up better than the one on which operation had been performed. The clarification of the cornea and the improvement of vision in my cases depended on the end-result of two different kinds of opacities, the one caused directly by the pannus and the other caused by the deposits of fat. The deposits of fat may disappear, but the improvement of vision may remain unsatisfactory, owing to the opacities of the pannus.

If one agrees with Ricker and considers the vascularization and the retardation of the blood stream to be the main causes for deposits of fat, one can assume that the resorption of the fat depends either on the obliteration or on the contraction of the blood vessels.

Thus the entire question may be reduced to this: To what extent is pannus capable of retrogression? In general, pannus is a progressive process. The condition of the cornea, the surface of which is entirely covered by blood vessels, may improve somewhat, but complete restoration is extremely rare. On the other hand, the course of pannus depends on the general condition of the organism. General etiologic factors, such as constitutional anomalies and tuberculosis, play an important part in the development of pannus. The trachomatous process of the conjunctiva is not more influenced by general conditions than any other chronic

21. Nakaizumi: Klin. Monatsbl. f. Augenh. **65**:760, 1920.

conjunctivitis or blepharitis. Pannus, however, is different. According to my observations the development of pannus is greatly influenced by the general condition of the organism, e. g., by disturbances of the endocrine system. Neither the local nor the general factors which cause pannus are alone sufficient to warrant deposits of fat. An additional factor is required, in my opinion, an increase in the fat content of the blood on a constitutional basis.

As far as therapy is concerned, I must emphasize the results of corneal transplantation. In my patients who were treated by this method the transplanted flap healed well and remained more or less transparent. One may ask whether a cornea with deposits of fat represents a favorable bed for the healing of a corneal graft. A confirmative answer to this question may be found in the following observation: It is known that the best results of free transplantation of the cornea are obtained in cases of leukoma resulting from interstitial keratitis. Here one is dealing with a condition analogous to pannus, in which there are an abundance of blood vessels and deposits of fat in the posterior layers of the cornea. It seems to me that the favorable results obtained by grafting in cases of a leukoma due to interstitial keratitis show a certain resemblance to the results in my cases of trachomatous pannus.

EXPERIMENTAL DETACHMENT OF THE RETINA

TREATMENT WITH SUBRETINAL INJECTIONS OF BLOOD PLASMA

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The purpose of this study was to discover whether blood plasma has any "gluing" effect when placed between the neuro-epithelial and the pigment layer of an experimentally detached retina, as claimed by Strampelli.[1] The method first employed to obtain detachment was a simple one, in which a spatula was used to separate the neuro-epithelial layer under observation through a hand ophthalmoscope. The difficulty of working on the fundus while watching the spatula through the ophthalmoscope, as well as the gossamer fragility of the tissue involved, made this method impracticable.

The final technic, and one which proved to be easy and satisfactory, was to bleb off the neuro-epithelial layer with physiologic solution of sodium chloride under direct observation through a hydrophthalmoscope.[2] The hydrophthalmoscope developed for working on the fundus consists of a brass chamber, *A*, with a small plano observation glass, *B*, set at an angle of 35 degrees to the scleral flange, *F*. A miniature 2 volt light, *E*, supplies illumination. In use the flange, *F*, rests on the sclera, where it makes an air-tight fit. A 10 inch (25.4 cm.) length of size 8 rubber catheter tubing is attached to the inlet, *D*, and its other end placed in a beaker of physiologic solution of sodium chloride. To the outlet, *C*, a similar piece of tubing is attached, and its distal end is connected with a 10 cc. syringe. The plunger of the syringe is then gradually withdrawn until the saline solution completely fills the brass chamber, when a bulldog clip is placed on the tubing from the beaker; a slight further withdrawal of the plunger of the syringe places a negative pressure in the device.

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1. Strampelli, B.: Treatment of Detachment of the Retina with Subretinal Injection of Blood Plasma, Boll. d'ocul. **12**:629-632, 1933.

2. Thomson, W. H. M.: The Hydrophthalmoscope, Canad. M. A. J. **32**:423-424, 1935.

A second clip placed on the tubing from the syringe seals the device so that it is securely fixed to the eye by negative pressure and takes the place of a fixation forceps. The corneal refraction is abolished

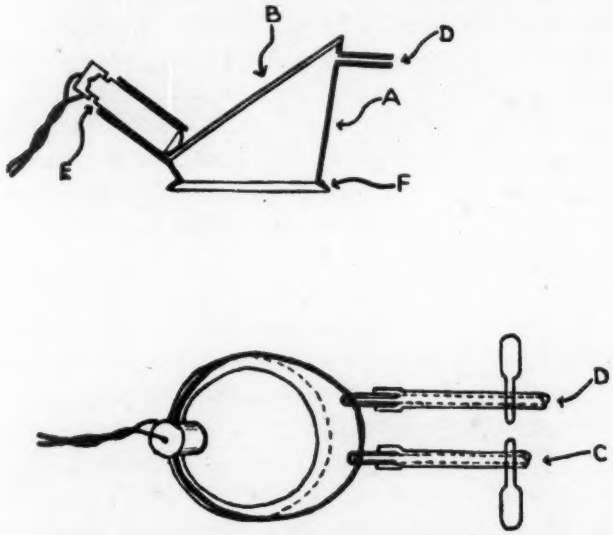

Fig. 1.—Two views of the hydrophthalmoscope.

Fig. 2.—Photomicrograph taken three weeks after the injection of blood plasma, showing strands of fibrin uniting the neuro-epithelial layer to the pigment layer.

by the saline solution, and the operator, with both hands free, can work with ease and precision on the neuro-epithelial layer. The angular placing of the observation glass, *B*, forms a prism and allows a view

of the fundus farther toward the ora serrata than would a flatly placed glass. The slight vacuum necessary for retention does not harm the corneal epithelium, and it has been left in place on a dog's eye for one hour without any clouding.

With this device in place, a 23 gage needle of sufficient length, with the point beveled to an angle of about 70 degrees, is now slowly inserted through the pigment layer and tapetum, and a slight pressure is made on the plunger of the syringe so that a little saline solution is forced under the neuro-epithelial layer to bleb it off. It is necessary to withdraw a small amount of vitreous through a 10 gage needle whenever a large detachment is required; otherwise the saline solution injected gives rise to glaucoma and immediate mistiness of the cornea.

Blood plasma previously obtained from the dog and kept from clotting by the use of ice or heparin is then injected under the neuro-epithelial layer after room is made for it by the withdrawal of a part of the saline solution used for detaching the retina.

Sections of eyes treated in this manner and enucleated from two weeks to a month later showed that minute strands of fibrin do form between the pigment layer and the neuro-epithelial layer and that the retina goes back into place (fig. 2). Saline solution alone when left between the retinal layers does not cause strands of fibrin to form, although the neuro-epithelial layer replaces itself in apposition to the pigment layer.

THE CORTICAL INNERVATION OF OCULAR MOVEMENTS

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While the cortical innervation of the skeletal muscles has been studied in much detail, there exists still much uncertainty about the cortical innervation of ocular muscles. Even the existence of such cortical centers was recently questioned by one of the most distinguished students of the problem (Muskens), contrary to the general opinion that assumes the existence of ocular centers in the frontal and occipital lobes since Ferrier's [1] classic experiments. Muskens [2] referred to stimulation experiments of Leyton and Sherrington [3] in the cortex of monkeys and apes showing that stronger stimuli are required to produce ocular movements than to produce responses of the skeletal muscles. Muskens was therefore inclined to presume that the eye movements observed in stimulation of the cerebral cortex are due to an escape of the current to the basal ganglions. He assumed that the lentiform nucleus is the site of origin of a centrifugal system innervating ocular movements. Paralysis of gaze in lesions of the internal capsule is explained, according to this theory, by encroachment of the lesion on the lentiform nucleus. The conduction of impulses to the nuclei of the ocular muscles is described by Muskens in the following way: The fibers originating in the globus pallidus of the lentiform nucleus descend to the nuclei of the posterior commissure. They are continued by fibers

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1. Ferrier: Proc. Roy. Soc. London **23**:409, 1875; Functions of the Brain, ed. 2, London, Smith, Elder & Co., 1886.

2. Muskens, L. J. J.: Monatschr. f. Psychiat. u. Neurol. **76**:268, 1930; Deutsche Ztschr. f. Nervenh. **115**:81, 1930; Das supravestibuläre System Amsterdam, N. V. Noord-Hollandsche Uitgeversm, 1934.

3. Leyton, A. S. F., and Sherrington, C. S.: Quart. J. Exper. Physiol. **11**:135, 1917.

that partially decussate in the posterior commissure, descend (in a caudal direction) in the posterior longitudinal fasciculus and finally end on the ocular nuclei.

There seems to exist a general agreement on one point at least; namely, the importance of the posterior longitudinal fasciculus for the conduction of impulses from the forebrain to the ocular muscles. This importance is shown by the observation that lesions of this bundle cause in man paralysis of gaze (Spitzer [4]) and prevent in animals (dogs) the appearance of horizontal conjugate movements of the eyes on cortical stimulation (Spiegel and Tokay [5]). The posterior longitudinal fasciculus contains two groups of fibers, ascending (running in a caudo-oral direction) and descending ones (running in an orocaudal direction). Considering the uncertainty in regard to the cortical innervation of the ocular muscles, it seems perhaps advisable to use this anatomic fact as a starting point and to study first the problem as to whether the ascending or the descending fibers of the posterior longitudinal fasciculus carry the impulses from the forebrain to the nuclei of the ocular muscles.

I. INNERVATION OF LATERAL MOVEMENTS: INVESTIGATION OF RÔLES OF ASCENDING AND DESCENDING FIBERS OF POSTERIOR LONGITUDINAL FASCICULUS AND OF VESTIBULAR NUCLEI

One of us (Spiegel) has already tried experimentally to approach this problem; he advised his pupil Dr. Ken Taga [6] to sever the posterior commissure and to study the effect of cortical stimulation on the eyes after this operation. It was shown in cats that destruction of the tectum mesencephali and of the dorsal wall of the sylvian aqueduct with the posterior commissure does not prevent the appearance of horizontal conjugate ocular movements on cortical stimulation. These experiments, however, informed us only about that part of the descending fibers of the posterior longitudinal fasciculus that reaches this bundle after decussating in the posterior commissure. It seemed necessary to carry out further experiments in order to ascertain, also, the rôle of those fibers that descend in the posterior longitudinal fasciculus without decussating in the posterior commissure. In order to reach all these fibers, it was necessary to injure the systems arising from the lentiform nucleus cranially to the posterior commissure and to study

4. Spitzer, A.: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **6**:1, 1899; **25**:423, 1924.

5. Spiegel, E. A., and Tokay, L.: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **32**:138, 1930.

6. Taga, K.: Arch. f. d. ges. Physiol. **223**:116, 1929.

the effect of such lesions on the ocular movements on stimulation of the cerebral cortex. One may injure these fibers in the place of origin, i. e., in the globus pallidus, or after they leave this ganglion. They form after perforating the internal capsule, medial to that system, the so-called Forel's field H_2 that is found dorsal to the corpus subthalamicum Luysii. In front of and dorsal to the red nucleus the fibers pass obliquely dorsomediad; most of them end, according to Morgan,[7] in the nucleus interstitialis Cajal, some in the nucleus of Darkschevitch in the posterior commissure. If one tries to reach the pallidofugal fibers from above, a puncture injuring them between the internal capsule and the posterior commissure has the advantage, in comparison with a puncture of the lentiform body, that the former lesion spares the internal capsule, while a puncture of the pallidum must pass through the internal capsule and may here affect corticofugal tracts to the ocular nuclei.

Method.—The experiments were performed on eleven cats. The lesions were made with Clarke's stereotaxic instrument. This had the advantage that only a small puncture of the cortex behind the sigmoid gyri was necessary, so that the cortical motor centers remained well excitable. The stimulation of the cortical centers for the eye was performed in some cases before as well as after injury of the pallidofugal fibers, in most experiments only after this operation, because the excitability of the motor cortex could be best preserved when this region was exposed as late as possible. In order to observe the eye movements the third lid was extirpated, an incision was made into the external canthus, and the lids were widely separated. The head was rigidly fixed during the cortical stimulation so that reflex movements of the eyes due to movements of the head were prevented. The extent of the lesions was controlled on serial sections stained by Weil's method.

Before we analyze the rôle of the pallidofugal system, it seems necessary to answer a preliminary question; namely, whether the eye movements observed on cortical stimulation are due to a spread of the current to subcortical ganglia or whether they represent true cortical reactions. This question was studied by comparing the threshold of cortical stimulation before and after an incision into the white matter below the stimulated cortical area. This incision severed the nerve tracts that connect this cortical area with the subcortex. After such an incision the separated cortical area was replaced in its original position and again stimulated. Motor effects that were then obtained by stimulation of this area were due only to an escape of the current to the cubcortex (fibers of the internal capsule or subcortical ganglions). Protocol 1 illustrates such an experiment.

The experiment shows that the threshold of ocular reaction was much higher after the incision into the white matter than before this

7. Morgan, L. O.: Corpus Striatum: Study of Secondary Degenerations Following Lesions in Man and of Symptoms and Acute Degenerations Following Experimental Lesions in Cats, Arch. Neurol. & Psychiat. **18**:495 (Oct.) 1927.

incision. As long as the corticofugal pathways were still intact, the stimuli sufficient to elicit ocular movement were distinctly weaker than those required when the ocular movements were produced by an escape of the current to the subcortex. It may be inferred that one can elicit ocular movements from the cortex directly, without an escape of the

PROTOCOL 1.—*Cat 9*

	Distance of Coils	Reactions of the Eyes	Other Reactions
		Stimulation of left Gyrus Sigmoideus Anterior with Bipolar Electrode with Cat Under Ether Anesthesia	
12:00	6.0 cm.	Both eyeballs to right and upward; dilatation of pupils	Cloni of right foreleg
	7.0 cm.	Both eyeballs to right and upward; dilatation of pupils	Cloni of right foreleg; general convulsions
	8.0 cm.	No reaction	No reaction
	7.5 cm.	No reaction	Slight outward rotation and flexion of right foreleg
12:04	8.0 cm.	No reaction	Inward rotation of right foreleg
12:05	8.0 cm.	Upward movement of both eyes	No reaction
12:07	7.0 cm.	Both eyeballs to right side; movement of right eye is more marked than that of left	Flexion of right foreleg precedes ocular movements
12:11	7.0 cm.	No reaction	No reaction
	6.0 cm.	Both eyeballs to right and upward	Cloni of right foreleg followed by short lasting cloni of right half of body
12:12	7.0 cm.	Right eye moves outward; both pupils remain slitlike	Tonic clonic convulsion of right foreleg
	7.50 cm.	Slow outward movement of right eye	Tonic flexion of right foreleg
	8.0 cm.	Slow conjugate movement to right and upward	Tonic flexion of right foreleg
	8.5 cm.	Right eye downward and somewhat outward	Inward rotation of right foreleg
12:15		Incision into white matter below gyri sigmoidei; gyri are replaced in former position	
12:28	7, 5, 3, 2 cm.	No reaction	No reaction
	1.0 cm.	Upward movement of both eyes; dilatation of pupils	General muscular movements
3:10	7 and 5 cm.	No reaction	No reaction
	4.0 cm.	Both eyes to right and downward	
		Stimulation of White Matter After Removal of Sigmoid Gyri	
	5.0 cm.	Both eyes to the right and slightly upward	
		Stimulation with Sigmoid Gyri Replaced	
	4.0 cm.	Both eyes upward	
	3.0 cm.	Both eyes to right side	

current to the subcortical systems. This refutes the assumption that the ocular movements observed on cortical stimulation are simply the result of the spreading of the current to the basal ganglions.

It remains, however, quite possible that the corticofugal impulses are not transmitted by long corticomesencephalic and corticorhombencephalic systems but by corticopallidal systems, and thence by pallidofugal tracts to the nuclei of the commissure. It seems therefore of interest to study the effect of severance of the pallidofugal systems on

the stimulation of the cortical centers of eyeball movements. Such experiments were made as follows:

Experiment on Cat 6.—A lesion was made with the Clarke apparatus on the right side; puncture 10, 9, 8 and 7 mm. anterior to the interaural line, 6 mm. outside the sagittal line. Stimulation of the right anterior sigmoidal gyrus yielded deviation of both eyeballs to the left side and dilatation of the pupils, as well as slight tonic movements of the left limbs. After the stimulation ceased, slow horizontal undulations of the eyeballs continued. A subsequent stimulation caused upward movement of the right eye and then divergence movements of the eyeballs. Finally conjugate deviation to the left and upward could be elicited; after stimulation the eyes returned slowly to the original position.

Fig. 1.—Incision (*I*) through the right half of the diencephalon lateral to the posterior commissure (*P C*) ending on the ventral surface medially to the cerebral peduncle.

Histologic Examination: The lesion traversed the optic thalamus laterally to the tractus habenulopeduncularis and on more caudal sections, through the posterior commissure, continued laterally to the central gray matter and this commissure (fig. 1). It reached the ventral surface medially to the cerebral peduncle. The fibers from H_2 to the posterior commissure were certainly severed.

Experiment on Cat 7.—The observations are given in protocol 2.

Histologic Examination: The lesion was located between the lentiform body (pallidum and putamen) and the internal capsule so that it severed the pallidofugal fibers (fig. 2). It reached the ventral surface of the hemisphere, encroaching on some of the most dorsal fibers of the optic nerve.

After lesions of the pallidofugal fibers to the nuclei of the posterior commissure, stimulation of the cortical ocular centers still yielded typical lateral ocular movements to the opposite side as well as vertical movements. In some cases convergence or divergence movements were also noticed. It was not necessary, in order to elicit the ocular movements after these lesions, to apply stronger stimuli than usual on anesthetized animals. One may infer that it is not necessary that the centrifugal fibers originating in the globus pallidus and connecting these ganglions with the posterior commissure remain intact for the transmission of corticofugal impulses to the ocular muscles. The same

PROTOCOL 2.—*Cat 7. Stimulation of Left Frontal Lobe with Cat Under Ether Anesthesia After Severance of the Pallidofugal Fibers*

	Distance of Coils	Reaction of Eyeballs	Other Reactions
11:44	6.0 cm.	Cloni downward; after stimulation ceases, conjugate deviation to left	General cloni
11:45	6.0 cm.	Convergence; after stimulation, conjugate deviation to left	
11:47	7.0 and 6.0 cm.	No definite reaction	Slight movement of right legs
	5.5 cm.	Conjugate deviation to right, followed by cloni; after stimulation, eyes return to left	
12:30		Electrolytic lesion on left side 8 mm. outside midline, 12 and 13 mm. cranial to and 7 mm. above interaural line	
1:30	5.5 cm.	Deviation of both eyes to right; dilatation of pupils	General convulsions
	6.0 cm.	No reaction	No reaction
1:40	5.5 cm.	Both eyes to right and upward	General convulsions
	6.0 cm.	No reaction	Slight movements of right foreleg
3:25	6.0 cm.	Both eyeballs upward; dilation of pupils	Slight flexion of right foreleg
3:29	5.5 cm.	Both eyeballs to right; pupils dilate	Slight movements of right forlegs
3:34	5.5 cm.	Both eyeballs downward, then to right (more marked on right eye)	
3:37	6.0 cm.	Both eyeballs upward	

applies of course to the descending fibers of the posterior longitudinal fasciculus that continue this pallidofugal system.

We must therefore take into consideration the rôle of the second part of the posterior longitudinal fasciculus—its ascending fibers. According to Cajal,[8] ascending fibers of the posterior longitudinal fasciculus originate in the vestibular nuclei, in the sensory nucleus of the trigeminus, in cells of the spinal cord and in cells of the reticular formation. Among these fibers only those from the vestibular nuclei are known to provide the connection necessary for conjugate lateral

8. Ramón y Cajal: Histologie du système nerveux, Paris, Maloine, 1911, vol. 2, p. 259.

ocular movements between the nuclei of the sixth and the third nerves. As to the fibers from the reticular cells, there enter the posterior longitudinal fasciculus axons that bifurcate in an ascending and descending branch, as shown by Cajal on silver impregnations. Experiments by Papez,[9] however, showed that after lesions of the reticular nuclei the degenerated fibers do not ascend for any distance above the lesion, while descending fibers could be followed as far as into the spinal cord. Thus the anatomy provides no foundation for the assumption that a so-called gaze center connecting the nuclei of the external and internal recti may be located in the reticular formation. The rôle of the ascending fibers originating in the vestibular nuclei was studied by Spiegel and Teschler [10] and by Spiegel.[11] These authors showed that bilateral

Fig 2.—Lesion (*L*) between the globus pallidus (*P*) and the internal capsule (*J*).

injuries to the vestibular nuclei changed the direction of the movements of the eyes elicited by stimulation of the frontal lobe as well as those elicited by stimulation of the occipital lobe. Conjugate deviation of the eyes to the side opposite the stimulated cortical areas depends, in particular, on the intactness of the vestibular nuclei. While normally horizontal deviation of the eyeballs prevails, after such injuries chiefly vertical movements were observed.

One might raise the objection that the changes in the direction of the eye movements following injuries to the vestibular nuclei are not consequential to the interruption of the corticofugal pathways to the

9. Papez, J. W.: J. Comp. Neurol. **41**:365, 1926.

10. Spiegel, E., and Teschler, L.: Arch. f. d. ges. Physiol. **222**:359, 1929.

11. Spiegel, E.: Rôle of Vestibular Nuclei in Cortical Innervation of Eye Muscles, Arch. Neurol. & Psychiat. **29**:1084 (May) 1933.

ocular nuclei but are perhaps due to concomitant lesions in the adjacent reticular substance or to shock of a neighboring gaze center (Bárány, de Nó) in these regions. Further experiments of Spiegel and Aronson [12] showed, however, that a change in the state of excitation of the vestibular nuclei without their being injured mechanically, for instance by unilateral labyrinthectomy, may also influence the effect of cortical stimulation. The interference of the spontaneous nystagmus after loss of one labyrinth, as well as of Bechterew's compensatory nystagmus with cortical impulses that tend to move the eyeballs in the opposite direction, eventually results in clonic eye jerks in the direction of the nystagmus, toward the stimulated hemisphere instead of toward the resting cerebral hemisphere.

If one compares the results of these experiments on the ascending part of the posterior longitudinal fasciculus and the results of lesions made in the pallidofugal system, as reported in this paper, the following may be inferred:

While injury of the ascending systems with the descending fibers left intact impairs or abolishes the lateral ocular movements on cortical stimulation, severance of the pallidofugal fibers that are continued by the descending system of the posterior longitudinal fasciculus has no noticeable effect on the cortical innervation of the ocular movements. No evidence was found that the pallidofugal system plays a part in the conduction of corticofugal impulses to the eye muscle nuclei, while the ascending fibers of the posterior longitudinal fasciculus, originating in the vestibular nuclei, play an important rôle in the innervation of lateral ocular movements of cortical origin.

II. INNERVATION OF VERTICAL MOVEMENTS

In view of the fact that injuries to the vestibular nuclei impair the horizontal ocular movements of cortical origin, while cortical stimulation still elicits vertical movements of the eyeballs, it seems necessary to devote a special study to the innervation of vertical movements. Ohm [13] expressed recently the belief, based on his clinical studies, that the vestibular nuclei transmit cortical impulses for lateral ocular movements as well as those for vertical movements, assuming that the preservation of the vertical movements in the aforementioned experiments of Spiegel may be due to preservation of a part of the vestibular nuclei. Another mechanism of vertical ocular movements was taken into consideration by Muskens.[14] He observed in pigeons that lesions

12. Spiegel, E., and Aronson, L.: Am. J. Physiol. **109**:694, 1934.

13. Ohm, J.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:136, 1935.

14. Muskens, L. J. J.: J. Comp. Neurol. **50**:289, 1930; Deutsche Ztschr. f. Nervenh. **115**:81, 1930. In his book (Das supravestibuläre System bei den Tieren und beim Menschen, Amsterdam, N. V. Noord-Hollandsche Uitgevers Maatsch app. j. 1934) he considers also the participation of the inferior olive in this mechanism.

of the median cerebellar nucleus are associated with forced movements of the eyes, head and trunk in the vertical plane, and he believes that the cerebellum, particularly the nucleus tecti, plays a rôle in the innervation of vertical ocular movements in quadrupeds also. We therefore studied in further experiments the influence of the destruction of the vermis cerebelli and extirpation of the whole cerebellum, respectively, and finally the effect of transverse section of the brain stem behind the midbrain on the cortical innervation of ocular movements.

Method.—The extirpation of the cerebellum was performed after opening the posterior atlanto-occipital ligament and removing the occipital bone as far as possible. The cerebellum was gradually elevated from the fossa rhomboidalis, and its brachia were severed. The extirpation of the organ was aided by the use of a suction pump connected with a small, slightly curved glass tube. The end of the tube was drawn into a point and could be easily introduced into all cor-

PROTOCOL 3.—*Cat 4. Extirpation of Cerebellum; Stimulation of Left Frontal Lobe*

	Distance of Coils	Reactions of Eyes	Other Reactions
4:50	6.0 cm.	Both eyes upward	
4:55	6.0 cm.	Bilateral up and down movements; after stimulation, both eyeballs deviated to right	General cloni
5:04	7.0 cm.	Right eye upward and inward; left eye upward (slow movements followed by vertical cloni)	General cloni
5:05	7.0 cm.	Tonic then clonic upward movements of both eyeballs, followed by cloni to left	
5:09	8.0 cm.	No effect	
	7.0 cm.	Slow movement of right eye upward and left eye downward, then bilateral vertical nystagmus	General cloni
5:11	7.5 cm.	Slow conjugate deviation downward and slightly to right; after cessation of stimulus, eyeballs move upward	No general reaction
5:14	7.0 cm.	Bilateral upward movement, then slow rhythmic movements to left	General cloni

ners of the posterior fossa, so that the remaining particles of the cerebellum could be easily removed by suction. Transverse section of the brain stem behind the midbrain was also performed from the posterior fossa after the fourth ventricle and the posterior aspect of the inferior colliculi were exposed. A knife bent in its plane at a right angle to the handle severed the cranial end of the fourth ventricle behind the midbrain.

Experiment on Cat 4.—Protocol 3 illustrates the effect of extirpation of the cerebellum.

Anatomic Examination: The cerebellum was almost completely extirpated. Only a small lobule of the flocculus remained on each side. These lobules were interspersed with hemorrhages. The floor of the fourth ventricle and the posterior aspect of the corpora quadrigemina posteriora were exposed (fig. 3).

Experiment on Cat 3.—Figure 4 shows the brain of cat 3; in this animal extirpation of the cerebellum (except the most lateral parts) was combined with transverse section of the cranial third of the pons. Stimulation of the left frontal lobe after this operation still elicited cloni of both eyes downward and then deviation of the left eye downward and of the right eye upward.

Experiment on Cat 11.—Protocol 4 illustrates the effect of extirpation of the vermis cerebelli and transverse section of the brain stem immediately behind the midbrain.

Anatomic Examination: The floor of the fourth ventricle was exposed. The transverse section of the brain stem lay immediately behind the inferior colliculus; it was complete with the exception of the lateral part of the left cerebral peduncle. All fibers ascending from the rhombencephalon to the cranial parts of the central nervous system were severed.

These experiments showed that extirpation of the cerebellum, including the nucleus tecti, and also severance of the brain stem behind the midbrain do not prevent the appearance of vertical ocular move-

Fig. 3.—Extirpation of the cerebellum (except the flocculi).

Fig. 4.—Transverse section through the cranial part of the pons; cerebellum extirpated (except the most lateral parts).

ments on cortical stimulation. Not only the vestibular nuclei and the nuclei tecti but also the whole rhombencephalon are dispensable in the cortical innervation of vertical movements of the eyeballs. We must therefore assume that the mechanism of this innervation differs from that of the lateral ocular movements; while the cortical innervation of the latter type of movements needs the ascending fibers of the posterior longitudinal fasciculus originating in the vestibular nuclei,

the connection of the forebrain with the midbrain suffices for the cortical innervation of the vertical movements. It is of course quite possible that neurons are intercalated between parts of the corticofugal tracts and the segmental nuclei of the ocular muscles innervating the vertical movements. The same applies to the connection between the cortex and the vestibular nuclei.[15] Further studies will be necessary to answer these questions.

SUMMARY

1. Conjugate ocular movements can be elicited by direct stimulation of the cortex without stimulation of the subcortex by an escape of the current.

PROTOCOL 4.—*Cat 11. Extirpation of Vermis Cerebelli, Exposing Floor of Fourth Ventricle; Transverse Section Immediately Behind Inferior Colliculi* *

Stimulation of Left Frontal Lobe			
Dura Closed	Distance of Coils	Reaction of Eyeballs	Other Reactions
12:05	5.0 cm.	Both downward, followed by vertical clonl; slight dilatation of pupils	
12:06	6.0 cm.	Upward movements of both eyes, with slight rotation of left eye	
12:10	Dura opened	No spontaneous oscillations of eyeballs	
	6.0 cm.	No reaction	
	5.5 cm.	Tonic-clonic upward movements	No reactions
12:15 and 12:20	5.5 cm.	Tonic-clonic upward movements	
12:25	6.0 cm.	Slow upward movement; no reaction of pupil	
12:26	6.0 cm.	Upward movement is more distinct in right eye; upward movement of left eye is combined with slight outward rotation	
12:28	6.0 cm.	Similar tonic reaction followed by clonl in same direction	
12:39	6.0 cm.	No reaction	
	5.5 cm.	Right eye upward; left eye first downward, then upward	
12:42	6.0 cm.	Tonic-clonic movement of both eyes upward	

* After this operation, the diameter of the pupils was 3 mm. The light reflex was preserved. The left frontal lobe was exposed. Slight undulation of the eyeballs in a vertical direction followed.

15. The supposition that neurons are intercalated between the corticofugal tracts and the vestibular nuclei was already expressed in former papers (e. g., Spiegel and Aronson [12]). Recent findings of W. Godlowski (Les centres sous-corticaux du regard et des mouvements associés des globes oculaires: Etude expérimentale, Krakow, 1936) showing by the degeneration method that fibers originating in the frontal lobe can be traced into the reticular formation of the pons but not as far as into the vestibular nuclei confirm this view. The conclusion of the author, however, that direct tracts from the reticular formation to the nuclei of the ocular muscles transmit the cortical impulses for lateral ocular movements cannot be accepted. Such a conception is contradictory to the fact that destruction of the posterior longitudinal fasciculus or of the vestibular nuclei abolishes the cortical innervation of lateral ocular movements. This fact indicates that cortical impulses for such movements, after reaching the reticulate substance of the pons, are thence transmitted to the vestibular nuclei and, by their neurons entering the posterior longitudinal fasciculus, to the nuclei of the ocular muscles.

2. Destruction of the pallidofugal system that is continued by descending fibers of the posterior longitudinal fasciculus does not prevent conjugate (lateral and vertical) ocular movements on cortical stimulation, while lesions of the vestibular nuclei impair or prevent cortical innervation of lateral conjugate ocular movements.

3. The cortical impulses for horizontal movements of the eyeball reach the nuclei of the ocular muscles by using ascending fibers of the posterior longitudinal bundles that originate in the vestibular nuclei, while the descending fibers of this bundle play no part in this innervation.

4. Extirpation of the cerebellum and transverse section of the brain stem behind the midbrain showed that the systems connecting the cerebral hemispheres with the midbrain suffice for the cortical innervation of the vertical ocular movements and that rhombencephalic neurons are dispensable in this mechanism.

ABSTRACT OF DISCUSSION

DR. ALFRED BIELSCHOWSKY, Hanover, N. H.: I want to congratulate Professor Spiegel and Dr. Scala on their excellent study. What is known about the motor innervation of the eyes is to be credited to the cooperation of physiologists, clinicians and anatomists. Physiologic observations showed that the individual ocular muscle is governed by different brain centers, according to whether it participates in a parallel or a nonparallel movement. This physiologic postulate has been confirmed by clinical experience. When the internal rectus muscles no longer cooperate with the external recti, the cause is a supranuclear lesion which injures the posterior longitudinal bundle between the third and the sixth nuclei. The normal function of the external rectus muscles excludes the possibility of a lesion injuring the pathway between the cortical centers and the sixth nuclei. Such a lesion will always cause loss of bilateral movement. Besides the internal recti there is only one pair of muscles in which one-sided paralysis can be attributed positively to a supranuclear lesion. In a patient with total paralysis of the right superior rectus muscle and almost complete paralysis of the left superior rectus muscle, the maximum impulse to look up raises the right visual line only to the horizontal position, whereas the left visual line is raised only a little above the horizontal plane. But if the patient is asked to close the eyes, and the lids are pulled open, the left eye moves up perfectly, whereas the right eye remains unmoved. The normal function of the left superior rectus muscle in Bell's phenomenon proves the integrity of the nuclei and roots of the nerves supplying the left superior rectus muscle, whereas the right oculomotor fibers are destroyed. The lesion that has cut off the left elevator nuclei from the cortical centers must be localized close to the nuclei, below the point where the pathway descending from the cortical center for the upward movement of the eyes bifurcates into the branches which go to both the third nuclei. One-sided elevator paralysis of supranuclear origin occurs extremely seldom. I have observed it only three times.

Associated paralyses of lateral movements display different signs and symptoms according to both the site and the size of the supranuclear

lesion. In typical cases the two eyes are equally restricted either in dextroversion or in levoversion, the convergent movement being intact. If the paralysis is caused by a lesion situated within the cerebral hemispheres, both head and eyes show a deviation of considerable magnitude toward the side of the lesion. An exact investigation is usually impossible, partly because of the bad mental condition of the patient, partly because of the quick recovery of the deficient ocular movement in cases in which death does not occur. The quick recovery is probably due to the fact that both hemispheres contain centers for dextroversion as well as levoversion, so that the center of the sound side begins to function vicariously. A patient with so-called pseudo-ophthalmoplegia is unable to move the eyes at the word of command, while if he is in a state of emotion or is interested in sensory excitations arising from outlying objects the eyes move involuntarily. If the patient's attention is directed to an object moving through the field of fixation, the eyes will follow in a fairly normal way. They react promptly to vestibular stimulations. Such a condition, which occurs in pseudobulbar paralysis, may be caused by a cortical lesion which injures the connections between the frontal oculomotor centers and other parts of the cortex. But a very similar behavior has been observed in diseases of the extrapyramidal motor system and of the corpus striatum; for instance, in paralysis agitans, pseudosclerosis, Wilson's disease and in postencephalitic parkinsonism. Differential diagnosis depends on the other neurologic signs and symptoms.

Paralyses of the associated parallel movements have been studied mostly in cases in which the lesion was situated within or near the pons and the region of the fourth ventricle, causing the loss of parallel lateral movement; and also, in cases of paralysis of the vertical movements in which the lesion could be localized in the region of the corpora quadrigemina or in the neighborhood of the sylvian aqueduct and the third ventricle respectively. At the height of the illness of a patient with associated paralysis of levoversion caused by acute encephalitis pontis, she had hemiparesis and hemianaesthesia dextra, paralysis of both facial nerves, associated paralysis of the ocular movement to the left and paresis of dextroversion. The convergence movement was undisturbed. The patient was unable to look to the left or at an object situated in the left half of the visual field. But when her head was turned with a sudden jerk to the right while she was fixing an object straight in front of her, the left eye moved outward almost to the normal limit, the right eye not quite as far inward. The same amount of levoversion could be performed if the object looked at was carried slowly to the left. The eyes remained directed to the left as long as the patient looked at the object, whereas in the first test, when the head was rotated with a sudden jerk to the right, immediately after the rotation the eyes slowly returned from levoversion to the original position without the patient being able to prevent it—a proof of the reflex character of the ocular movement elicited by a vestibular stimulation. The following movement to the left produced by the other test must be of cortical origin, because the patient was able to keep her eyes in levoversion as long as her attention was directed to the object the movement of which the eyes had followed. That this assumption is correct had been proved in other cases in which at first only the voluntary movement,

in a later stage also the following movement, was lost, whereas the eyes still responded to vestibular stimulations. Finally, even these were unsuccessful. Postmortem examination showed a tumor infiltrating the pons. In an example of associated paralysis of the superior rectus muscles the eyes can be raised voluntarily or at word of command only to the horizontal plane and cannot pass the latter even if the fixed object is moved slowly from below the horizontal plane upward or if the head is rotated downward with a sudden jerk. But a maximum elevation can be obtained in Bell's phenomenon—an irrefutable proof of the supranuclear site of the lesion and the intactness of the nuclei of the superior rectus muscles. A tumor of the corpora quadrigemina was diagnosed and was found on operation. At first, after removal of the tumor, the superior rectus muscles began to react to vestibular stimulations; the next faculty to return was that of following an object which was slowly moved upward; the last was that of elevation at the word of command. When, in addition to the aforementioned signs and symptoms of supranuclear associated paralyses, diplopia due to a paralytic squint arises, one must infer that the lesion has spread, involving the nuclei and the roots, respectively, of the motor nerves. A variation in the mode and the amount of action of the paralyzed muscles according to the method of stimulation will prove that the disorder is caused partly by a supranuclear, partly by a peripheral, lesion.

From the variety of signs and symptoms found in cases of associated ocular paralyses, one may draw the conclusion that ocular movements are to be divided into three groups:

To the first group belong the voluntary movements and the movements at command. They are governed by the cortical centers located in the frontal lobe.

The second group comprises the so-called psycho-optic reflex movements, which are produced by sensorial stimuli but are formed more or less instinctively. To this group belong the fusion as well as the attraction movements, the latter taking place if an object situated in the periphery of the visual field attracts attention. There are sufficient reasons for placing the cortical centers of those movements in the occipital lobes. Separate centers and pathways have been assumed by some authors because of the cases in which the attraction movement but not the so-called following movements are lost. There is no need for this hypothesis. The following movement may be produced in the same way as fusion movements that are independent of the will. From testing the vertical duction power, for instance, it is known that, as a rule, a person is unable in the beginning to overcome a prism of 5 degrees or more. But by beginning with a prism of 1 degree and gradually increasing the strength of the prism one can induce him finally to overcome 8 degrees or more by the appropriate vertical divergence. The nearer the image looked at is to the fovea, the stronger is the motor excitation that is produced. If a weak prism is placed base out before one eye, adduction takes place involuntarily, whereas in spite of causing diplopia a strong prism in the same position does not produce that movement. If the pathway descending from the occipital center is injured but not interrupted, the patient may be able to perform only a very small attraction movement induced by the excitation of paracentral retinal points. When the fixed object is moved very slowly so that its

retinal images move from the foveas to places in its proximate vicinity, a small compensating movement is required to bring the images back to the foveas. From the summation of such small innervations a movement of considerable extent can finally result.

The ocular movements of the third group are true reflex movements, elicited either by Bárány tests or by stimulation of the vestibular apparatus by sudden changes of posture of the head and body. These reflex innervations are transmitted to the motor nuclei of the eyes by the posterior longitudinal bundle.

The careful study of the signs and symptoms of ocular paralyses is of great importance, first, for neurologic diagnosis, because they are rather frequently the earliest and for a considerable period the only reliable proofs of organic intracranial disease; they may give, furthermore, very valuable clues for an exact localization of the lesion. They confirm or disprove, as the case may be, theories founded on physiologic investigations and experiments. The different symptoms produced by lesions of different size and localization hold many unsolved problems concerning structure and functioning of the nervous system, in the solution of which physiologists, anatomists and pathologists may join the clinicians.

It gives me great pleasure to acknowledge that the results of the excellent work done by Scala and Spiegel fully agree with the results of my clinical studies on the associated paralyses of the eyes.

Dr. Ernest A. Spiegel, Philadelphia: We are fortunate to have, to discuss our paper, a man who is the best clinical expert in the field of neuro-ophthalmology. I wish to express my sincere thanks to Professor Bielschowsky for his valuable analysis of these clinical symptoms. As he has pointed out, there is a group of clinical cases in which one cannot assume that the paralysis is due to lesions of the nuclei and one must assume that it is due to a lesion of a supranuclear center. The task of the pathologists therefore has been, for many years, to find such a center. A study of the literature on the subject will show that there are many theories concerning this point. Our systematic studies, however, showed that the assumption of such centers in the pallidum, the thalamus, the tectum mesencephali or the reticulate substance of the rhombencephalon is quite hypothetic. The importance of the vestibular nuclei could also be shown by cortical stimulation after unilateral labyrinthectomy (experiments with Dr. Aronson). If one stimulates, for instance, the right cortical centers of ocular movements after left-sided labyrinthectomy one finds in the brain two opposing forces, one force from the labyrinth nuclei, tending to produce nystagmus to the right side, and an opposing force from the right cortex, tending to produce it to the left side; if one now stimulates the cortex, the result is that the cortical force is stopped and the vestibular impulse determines the direction of the eye movement. The occurrence of pseudo-ophthalmoplegia in diseases of the extrapyramidal centers mentioned by Professor Bielschowsky is probably due to encroachment on corticofugal fibers, since pyramidal symptoms also may be found in such cases.

TESTS FOR HETEROPHORIA: A PROTEST

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Two of the most eminent authorities on muscle imbalance of the last half-century, Alexander Duane and Ernest E. Maddox, have enunciated the essential conditions of a satisfactory test for the muscle balance. Duane was the most strenuous advocate of the objective cover test with its subjective complement, the parallax test. Maddox, in referring to this cover test in an introductory address on heterophoria before the Ophthalmological Society of the United Kingdom, 1929, said that the most important thing was to keep the eye "covered long enough for the deviation to develop." The prolonged occlusion test is a combination of these two conditions. It puts into practice the principles which these two authorities have preached. It seems doubtful, however, whether either of them put the combined principles into practice and carried out an actual prolonged test. Indeed, it is doubtful whether Maddox realized the full significance of his dictum. Two other conditions, however, are necessary to obtain the full benefit of the test and to enable a reliable estimate of its value to be made.

In the first place, the test must be carried out with meticulous care as to detail, and, in the second place, there must be an intelligent interpretation of it. These conditions have often been lacking and in various ways. Thus, the duration of the test has not been long enough. For instance, in 20 of 29 cases reported in one paper the test lasted from one to twenty-four hours. This is not the prolonged occlusion test. In one case the patient was told to remove the glasses when she came into the waiting room, and the test was thereby rendered valueless. In another instance the patient was told to wear the glasses several hours a day, the result being that the oculist "did not think much of the test." In another case the patient did not put on his glasses until after he was dressed. In another instance a patient was told to wear the ground glass for four days and then return, no instructions being given as to the necessity for continuity. These are some of the ways in which the value of the test is vitiated or destroyed. Presumably this lack of attention to detail explains the statement in Berens' recent book, "The Eye and Its Diseases," that the test is *sometimes* of value and in other cases induces artificial conditions. This statement apparently represents the prevailing opinion and not that of any one person in particular and therefore will be commented on in a purely impersonal way.

It must be assumed, however, that it is acceptable to the author (L. C. Peter) and the editor (C. Berens).

It is interesting and instructive to note the various ways in which the adoption of the Duane-Maddox principles modifies the findings and therefore the conclusions and treatment.

My own impression, based on the observation of more than 2,000 cases, is that the occlusion test is *always* of value, its value varying, of course, in different cases. Thus, if orthophoria is found at the end of the period of occlusion it is fairly (but not absolutely) safe to say that muscle imbalance as a cause of the symptoms is practically eliminated, and this finding is of value, making it necessary to seek the cause in some other direction. In many cases the amount of latent error is surprising, as are also the variations from the results obtained at the preocclusion test. In cases of hyperphoria one finds a great difference in frequency as shown by the ordinary short tests and the prolonged test, the ordinary tests giving the manifest or functional phoria and the prolonged occlusion test the latent phoria or a close approximation to the total amount. By the ordinary methods the percentage of hyperphoria cited in this article (Peter) varied from 7 to 35 per cent. In a study of 700 cases Hansell and Reber found hyperphoria of 0.5 degree or more in 20 per cent. Among 700 cases in which the prolonged occlusion test was given in which one eye only was occluded, hyperphoria occurred in 84 per cent. In another series of 82 cases in which each eye was occluded, hyperphoria occurred in 98 per cent. This is a striking difference and means that a great many cases of hyperphoria must be overlooked with the ordinary methods of testing. When the Duane-Maddox principles are applied, it is found in cases of pure hyperphoria (as determined by the preocclusion test) that exophoria also (up to 13 degrees) is present in 77 per cent and esophoria in 8 per cent. Moreover, in an appreciable number of cases the hyperphoria changes to the opposite kind. These conditions account for the failure to obtain relief in many cases when the occlusion test is not carried out. The case of Mr. S. is not an uncommon one, the true character of which would surely be missed by the ordinary methods. The patient had severe sick headaches and neurasthenia. The ordinary tests showed orthophoria. Occlusion of the left eye for eight days caused no change, orthophoria still being maintained. The right eye was then occluded, and hyperphoria of 2 degrees developed in this eye, partial prismatic correction of which gave the desired relief. This was a case of reversion to an evolutionary period when the two eyes were working independently of one another—a monocular imbalance. It is unnecessary to say that these considerations call for a radical modification of the methods of dealing with muscle imbalance. If the diagnosis and treatment are

based solely on the manifestation of hyperphoria by the ordinary tests, a great many cases of latent heterophoria will be overlooked. Thus, among 104 cases of pure exophoria which were detected by the ordinary tests, hyperphoria developed in 82 per cent. It is obvious, therefore, that hyperphoria cannot be dealt with efficiently by the short tests detailed in this article (Peter), which show the manifest error only.

In dealing with esophoria the first question is one of diagnosis. What is the ophthalmologist really dealing with? It is well known that in some cases esophoria is due to accommodative spasm and is eliminated by the correction of the refraction. These are cases of secondary or pseudo-esophoria. Other cases of esophoria cannot be recognized as instances of true esophoria unless the eyes are examined on Duane-Maddox principles. Then one finds some curious revelations. Among 55 cases of pure esophoria detected by the preocclusion test, the esophoria was increased in 12 per cent (from 1 to 12 degrees), decreased in 38 per cent (from 1 to 6 degrees) and changed to exophoria in 38 per cent (from 1 to 10 degrees), and hyperphoria developed in 77 per cent of the cases. Thus it is evident that in cases of manifest pure esophoria the appearances are misleading. In cases of esophoria associated with hyperphoria the same holds true. Thus, among 78 cases of hyperesophoria, the esophoria diminished in 40 per cent and changed to exophoria (from 3 to 10 degrees) in 42 per cent, and hyperphoria increased (from 0.50 to 4 degrees) in 30 per cent and was reversed in 20 per cent. It is obvious that the determination of the manifest esophoria by short tests is *not* a sufficient preparation for dealing with this condition. Before eliminating the use of tea, coffee, tobacco, etc., it would be in the patient's interest to subject him to an occlusion test. Other comments might be made, but these suffice to indicate the importance of the practical application of the Duane-Maddox principles in cases of this type.

In exophoria the same general criticisms apply. In the first place, in many cases the condition cannot be recognized as exophoria unless an occlusion test is carried out. Thus, among 154 cases in which orthophoria was present at the time of the preocclusion test, exophoria (up to 16 degrees) developed in 76 per cent, either alone or in association with hyperphoria. In other words, no exophoria could be detected by short tests. On the other hand, among 104 cases of uncomplicated exophoria, the exophoria increased (from 1 to 16 degrees) in 91 per cent, and hyperphoria (from 0.50 to 4 degrees) developed in 82 per cent. In other words, hyperphoria was missed in 82 per cent of the cases by these short tests and the degree of exophoria underestimated. The author (Peter) laid stress on the importance of determining the amount of abduction and adduction. Adduction is too variable to be of any

value, and the prolonged occlusion test shows beyond any doubt that there is no constant relation between the preocclusion abduction and the amount of exophoria actually present.

It will be obvious from the foregoing remarks that I regard the statement in Peter's article on heterophoria, namely, that prolonged occlusion of one eye is "of value in *some* instances and creates an *artificial disturbance* in others," as falling far short of recognizing the true value or significance of this test. My impression is that the ordinary short tests described by Peter in Berens' book give simply the manifest error or functional balance, whereas the prolonged occlusion test gives the latent error or a close approximation to it and consequently is always of value, whether it furnishes the complete solution of the problem or not, and that it does not create artificial disturbances. It would be more accurate to say that it eliminates them. Failures are due to the faulty way in which the test is carried out and interpreted.

CARNOSINE OF THE OCULAR TISSUES

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Since the normal biochemistry of the ocular tissues is practically unknown, it is difficult to determine the course of events leading to pathologic changes. One method of studying the biochemistry of a tissue is to analyze the tissues and to determine quantitatively the amounts of the various major constituents. In a previous series of articles,[1] too numerous to mention here, the results of the analyses of two major constituents of the ocular tissues, i. e., the proteins and the lipids, were reported. During the past two years interest was turned toward an inquiry into the nature of the third constituent, the water-soluble extractives. In most of the ocular tissues these substances occur in minute quantities and are difficult to isolate and identify chemically. Frequently the water-soluble extractives can be determined only quantitatively, one at a time, in the tissues. Consequently the investigation may be limited to a few substances.

This article is a report on the amount of two water-soluble nitrogenous bases in the ocular tissues. They are the dipeptides anserine and carnosine.

METHOD

The tissues were immediately dissected from one hundred fresh ox eyes, comminuted and then extracted three times for one hour with about 5 volumes of water at 98 C. The combined extracts were heated to the boiling point and acidified with sufficient acetic acid to give a maximal precipitation of the proteins. After the extract was chilled, to solidify the fat, it was filtered, and the precipitate was washed with cold water. The extract and wash water were then heated on a water bath, evaporated to a small volume in a current of air and poured into 4 volumes of 95 per cent ethyl alcohol. After twenty-four hours the precipitate was centrifugated off, and the solution was treated with a mixture consisting of 25 per cent mercuric sulfate dissolved in 5 per cent concentrated sulfuric acid as long as a white flocculent precipitate formed. After standing in the cold a few hours the solution was centrifugated. The precipitate was suspended in water and decomposed with hydrogen sulfide. The mercuric sulfide was removed and washed with hot hydrogen sulfide water. The solution and washings were combined and the hydrogen sulfide removed. Barium hydroxide was added to the solution until it was slightly acid (p_H 5.0). The barium sulfate was removed,

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1. Krause, A. C.: The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934.

and the solution was evaporated to a small volume. Carnosine was determined on an aliquot sample according to a modified procedure [2] of the method of Koessler and Hanke.[3]

The dipeptides of another aliquot sample were hydrolyzed by heating them with a 20 per cent solution of hydrochloric acid in boiling water for six hours. After the proper corrections were made the amount of anserine was estimated by calculation from the colorimetric value of carnosine and from the amount of aminonitrogen as determined by Van Slyke's method before and after hydrolysis.[4]

The quantity of amino-acid nitrogen given off before and after hydrolysis corresponded to the amount of carnosine as determined by the colorimetric procedure on the extracts of the ocular tissues with the exception of those of lens, vitreous humor and retina. In these tissues the results obtained by applying the method of Wilson and Wolff [4] suggested that anserine was present and was reported erroneously as present in the bovine retina by me.[1] In the final mercuric sulfate extract of the lens, however, it was found, for example, in one analysis that the total amino-acid nitrogen content after hydrolysis, 2.7 mg., was equivalent to 2.4 mg. of aminonitrogen of hydrolyzed oxidized glutathione and to 0.3 mg. of aminonitrogen of carnosine as determined by the colorimetric method. In the hydrolyzed solution the quantity of cystine was found by the method of Folin and Marenzi [5] to be equivalent to the amount of the glutathione. Therefore, it was concluded that no anserine was present in the lens. The glutathione in the vitreous humor behaved in the same manner. Furthermore, no evidence was obtained for the presence of lenticular or retinal anserine in the lysine fraction after the use of the silver nitrate and barium hydroxide method of separation on the extract of 2 Kg. of lenses and 0.5 Kg. of retina.

Carnosine was isolated in small quantities as the blue copper salt from each of the ocular tissues except those of the cornea and sclera. No attempt was made to prepare the salt from these tissues. The carnosine showed a color value and a value for amino-acid nitrogen after hydrolysis which were equivalent to about 95 to 100 per cent carnosine. The decomposition point of the copper salt of lenticular carnosine was 214 C. (corrected), corresponding to that of the same salt isolated from the external ocular muscles.

In fresh brains of adult cattle which were treated in the same manner as the retinas, 7 and 7.2 mg. of carnosine per hundred grams was found. The copper salt decomposed at 211 C. (corrected) and gave off aminonitrogen before and after hydrolysis, corresponding to carnosine. No anserine was obtained from 20 Kg. of brain.

COMMENT

Carnosine, which is also called beta-alanylhistidine, was first isolated in 1900 from beef extract.[6] Anserine (methylcarnosine) was discovered

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3. Koessler, K. K., and Hanke, M. T.: A Microchemical Colorimetric Method for Estimating Imidazole Derivatives, J. Biol. Chem. **39**:497, 1919.

4. Wilson, D. W., and Wolff, W. A.: The Determination of Anserine in Muscle, J. Biol. Chem. **100**:cvi, 1933.

5. Folin, O., and Marenzi, A. D.: Tyrosine and Triptophane Determinations in One-Tenth Gram of Protein, J. Biol. Chem. **83**:89, 1929.

6. Gulewitsch, W., and Amiradžibi, S.: Zur Kenntnis der Extractivstoffe der Muskeln, Ztschr. f. physiol. Chem. **30**:565, 1900.

in 1929 in goose muscle.[7] Later carnosine was found to occur together with anserine in the muscle of certain vertebrates.[8] In the muscle of the ox and horse, however, carnosine was found to exist alone. In certain lower vertebrates, such as the codfish, anserine but no carnosine was detectable. In contrast to vertebrate muscle, the invertebrate muscle was found to contain arginine, which apparently takes the place of creatine and carnosine. Prior to this report neither anserine nor carnosine was found in the eye tissues of animals.

Anserine and carnosine presumably play an important rôle in the specific function of certain tissues. They occur in relatively high concentration, possess an imidazole structure and contain the only naturally occurring group of beta-amino-acids. They are not easily hydrolyzed by extracts of ocular or other tissues. Evidently carnosine and presumably anserine are retained in the cells and are not diffusible, at least in the

Carnosine of the Ocular Tissues in Milligrams per Hundred Grams

Sclera		Cornea: Whole		Cornea: Epithelium		Cornea: Without Epithelium		Choroid		Iris		Optic Nerve		Retina		Vitreous Humor		Lens		C jun
Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet
1.94	6.9	4.36	22.6	17.8	89.6	2.43	12.3	16.8	60.4	3.2	16.8	1.47	6.0	2.6	21.4	0.16	11.3	4.7	13.4	11.0
2.18	6.8	4.62	23.4	18.6	90.6	2.22	11.6	16.9	68.4	3.7	19.6	1.54	7.3	2.6	20.8	0.29	17.2	5.7	16.6	11.3
1.7	6.2	4.67	22.4	18.3	93.3	2.63	12.4	17.8	73.4	4.0	22.3	1.9	7.2	3.0	25.2	0.27	16.4	5.2	14.9	9.9
2.43	9.88	4.54	21.8	17.2	89.0	2.00	11.0	16.1	63.8	4.1	20.1	1.7	6.6	2.8	22.7	0.23	15.8	5.7	17.1	10.0
2.61	9.04			17.2	89.0	2.30	11.4					1.8	7.2	3.0	24.2			5.5	14.6	
														2.34	17.82					
														2.66	21.8					
														2.74	22.5					

muscle cells.[2] The administration of large doses of carnosine produces no toxic symptoms.[9] From these few facts, however, there is no experimental evidence that points to a definite physiologic activity.

An interesting theory to explain the formation of carnosine with its beta-alanine group was proposed by Blanchetière.[10] Since carnosine cannot arise from alpha-alanine, he suggested that an initial dipeptide of histidine and aspartic acid condenses to form an internal anhydride which is capable of hydrolyzing into two different peptides. One of these

7. Ackermann, D.; Timpe, O., and Poller, K.: Ueber das Anserin, einen neuen Bestandteil der Vogelmuskulatur, Ztschr. f. physiol. Chem. **183**:1, 1929.

8. Wolff, W. A., and Wilson, D. W.: Anserine in Mammalian Skeletal Muscle, J. Biol. Chem. **95**:495, 1932; Carnosine and Anserine in Mammalian Skeletal Muscle, ibid. **109**:565, 1935.

9. Rasenkow, I. P.; Derweis, G. W., and Ssewerin, S. E.: Zur Frage nach Carnosinwirkung auf die Magensaftsekretion, Ztschr. f. physiol. Chem. **162**:95, 1926.

10. Blanchetière, M. A.: Constitution des anhydrides des acides aspartique et glutamique; son importance biologique, Bull. Soc. chim. biol. **6**:854, 1924.

peptides belongs to the group of very reactive peptides which lose carbon dioxide easily. In this case the more reactive peptides form carnosine. The possible occurrence of histidine and aspartic acid in the water-soluble extractives of ocular tissues will be investigated later in this laboratory.

In the ocular tissues carnosine shows a wide distribution in low concentrations (see the table). There is apparently no relation of tissue function or structure to the amount of this substance. The sclera with its fibrous tissue poor in nuclei and the retinal nerve tissue with its many nuclei contain about the same quantity. The greatest difference in the ocular tissues exists between the epithelium of the cornea and the remaining corneal structure. The richly vascularized choroid possesses about three times as much carnosine as the avascular lens. Carnosine also occurs in small amounts in the vitreous humor, which has no cells and no blood supply. It is to be expected that carnosine would be found in the muscular iris, because it occurs in skeletal muscle. There seems to be no particular physiologic basis for the distribution of carnosine in the ocular tissues.

SUMMARY

The distribution of carnosine in the bovine ocular tissues was determined. No anserine was found.

Many of the technical procedures were performed by Mr. Warren Tauber.

STUDIES IN HISTOCHEMISTRY

X. DISTRIBUTION OF VITAMIN C IN THE LENS OF THE EYE

DAVID GLICK, PH.D.
AND
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SAN FRANCISCO

The lens of the eye, having no blood supply, is especially dependent on those intracellular substances which form oxidation-reduction systems for the maintenance of many of its metabolic processes. In this connection, a great deal of work has been published recently on the occurrence and rôle of vitamin C in the lens.

The concentration of vitamin C in the lens as reported for a number of animals is shown in table 1.

Apparently no significance can be attached to the concentration of vitamin C in the lens in regard to the capacity of some animals, such as the rat, rabbit, sheep, ox, cow and horse, for synthesizing this vitamin and the lack of this capacity in animals such as the guinea-pig and man, who must obtain vitamin C from their food. The variation in the concentration of vitamin C between these two groups is less than the variation within the groups. Thus the very low value for the rat lens stands in striking contrast to the much higher values for the lenses of the other animals of the group capable of synthesizing the vitamin.

The important observation has been made repeatedly that the concentration of vitamin C falls in proportion to the clouding in cataractous lenses, to values approaching zero.[1] Also the concentration of vitamin C in the lens decreases with advancing age.[1c] Vitamin C seems to be

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From the Pathological and Research Laboratories of the Mount Zion Hospital, San Francisco.

1. (*a*) Birch, T. W., and Dann, W. J.: Estimation and Distribution of Ascorbic Acid (Vitamin C) and Glutathione in Animal Tissues, Nature, London **131**:469 (April 1) 1933. (*b*) von Euler, H., and Malmberg, M.: Ueber die antiskorbutische Wirkung der Augenlinsen und über ihren Gehalt an Reduktonen und Sulfhydrylen, Ztschr. f. physiol. Chem. **230**:225, 1934. (*c*) Müller, H. K.: Buschke, W.; Gurewitsch, A., and Brühl, F.: Vitamin C in Kammerwasser und Linse—Seine Bedeutung für Physiologie und Pathologie des Linsenstoffwechsels, Klin. Wchnschr. **13**:20 (Jan. 6) 1934. (*d*) von Euler, H., and Malmberg, M.: Neue Versuche über Ascorbinsäure (C Vitamin) in tierischen Augenlinsen, Arch. f. Augenh. **109**:225, 1935. (*e*) Weinstein, P.: Rôle of Vitamin C in Biologic Oxidation of Crystalline Lens, Orvosi hetil. **79**:874 (Aug. 10) 1935.

important for the respiration of the lens tissue, and the drop in concentration associated with cataract formation and increasing age is paralleled by a drop in the rate of respiration.[1c]

Von Euler and Malmberg [1b] observed no change after forty-eight hours in the concentration of vitamin C in the rabbit lens when 20 Gm. of dinitrophenol or 1 Gm. of naphthalene was administered by mouth. Later the same authors [1d] reported that they could not produce cataracts in rabbits even when 0.5 Gm. of naphthalene was given daily for fifty-seven days. But Müller and his coworkers [1c] reported that they had

TABLE 1.—*Concentration of Vitamin C in the Lenses of Various Animals*

Animal	Mg. of Vitamin C per Gm. of Tissue	Reference
Sheep	0.46	Birch and Dann [1a]
Ox	0.26-0.40	von Euler and Martius: Ztschr. f. physiol. Chem. **222**: 65, 1933
Guinea-pig	0.16	von Euler and Malmberg [1d]
	0.075	Müller and Buschke: Schweiz. med. Wchnschr. **15**: 585 (June 23) 1934
Rabbit	0.16	von Euler and Balmberg [1d]
	0.07-0.17	Müller, Buschke, Gurewitsch and Brühl [1c]
	0.21	Müller and Buschke: Schweiz. med. Wchnschr. **15**: 585 (June 23) 1934
Fish	0.32-1.04	von Euler and Malmberg [1d]
Rat	0.02	von Euler and Malmberg [1d]
	0.027	Müller and Buschke: Schweiz. med. Wchnschr. **15**: 585 (June 23) 1934
Man	0.31	von Euler and Malmberg [1d]
Cattle	0.30-0.40	Müller, Buschke, Gurewitsch and Brühl [1c]
	0.335	Müller and Buschke: Schweiz. med. Wchnschr. **15**: 585 (June 23) 1934
Horse	0.39	Müller and Buschke: Schweiz. med. Wchnschr. **15**: 585 (June 23) 1934

produced naphthalene cataracts in rabbits, and though there was no change in the concentration of vitamin C in the lenses at first, the value fell to as low as zero as they later became cloudier. These cataracts could be prevented by feeding cabbage, according to Bourne,[2] but Müller and Buschke [3] obtained inconclusive results through treatment of naphthalene cataracts with vitamin C. Recently Josephson [4] claimed to have

2. Bourne, M. C.: Effect of Diet on Nature of Ocular Lesions Produced by Naphthalene, Brit. J. Ophth. **17**:210 (April) 1933.

3. Müller, H. K., and Buschke, W.: Ueber die Behandlung der Naphthalinkatarakt mit Vitamin C, Arch. f. Augenh. **108**:597, 1934.

4. Josephson, E. M.: Ascorbic Acid in Cataract with Special Reference to Dinitrophenol Cataracts, Science **82**:222 (Sept. 6) 1935.

produced marked improvement in lenses of patients with cataract, including those with cataracts due to dinitrophenol, by the daily administration of from 15 to 300 mg. of vitamin C. This finding awaits further elaboration and confirmation.

From the foregoing review of some of the work constituting the background of the study of vitamin C in the lens it would appear desirable to know something of the distribution of this important substance in the various regions of the lens. The lens is not a homogeneous body. Its central portion or nucleus contains a higher percentage of solid matter, is more rigid and has a greater specific gravity and a higher refractive index than the outer portions.[5] Though supporting data were not included, the statement has been made by Müller and his co-workers [1c] that the nucleus of the lens contains a smaller concentration of vitamin C and has a lower rate of respiration than the outer regions.

It was the purpose in the investigation here reported to study quantitatively the concentration of vitamin C from the periphery of the lens to its center. This study has been made possible by application of the Linderstrøm-Lang and Holter microtitration apparatus,[6] which enables determination of the concentration of vitamin C in microtome sections of tissue.[7]

EXPERIMENTAL METHODS

As in our previous investigations in histochemistry, we employed bovine tissues. Shortly after the animals were killed the lenses of the eyes were removed and frozen hard at —5 C. Sampling of tissue was performed by pushing a cork borer having an internal diameter of 4.2 mm. through the center of the frozen lens perpendicular to the plane of the greatest circumference. The cylinder of tissue thus obtained was placed on a rotary freezing microtome, and sections 30 microns thick were made. Every twentieth section was placed in 50 cu. mm. of a 2 per cent solution of sulfosalicylic acid contained in a titration tube, and the titration of the vitamin C was carried out with a solution of 2, 6 dichlorophenolindophenol, each cubic centimeter of which was equivalent to 0.13 mg. of vitamin C.[7] The solution of sulfosalicylic acid was used instead of the acetic acid previously employed since the possibility of interference with the titration, due to the presence of glutathione, is thus eliminated. Cysteine may still interfere, however.[8]

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5. Mathews, A. P.: Physiological Chemistry, ed. 5, Baltimore, William Wood & Company, 1931, p. 716.

6. Linderstrøm-Lang, K., and Holter, H.: Enzymatische Histochemie, in Nord, F. F., and Weidenhagen, R.: Ergebnisse der Enzymforschung, Leipzig, Akademische Verlagsgesellschaft m. b. H., 1934, vol. 3, p. 309. Glick, D.: Methods and Applications of Enzyme Studies in Histological Chemistry by the Linderstrøm-Lang Holter Technic, J. Chem. Education **12**:253 (June) 1935.

7. Glick, D.: The Chemical Determination of Minute Quantities of Vitamin C, J. Biol. Chem. **109**:433 (April) 1935.

8. According to a private communication from Dr. H. Borsook, California Institute of Technology, Pasadena.

By the aforementioned procedure the quantity of vitamin C per section of known volume was determined, but in order to obtain the concentration of vitamin C in the tissue it was necessary to determine the specific gravity. Since the specific gravity increases toward the center of the lens, measurements of specific gravity must be made on a series of microtome sections so that a correlation may be drawn between the quantity of vitamin C in a given section and the specific gravity at the site of this same section.

Several methods were tried to determine the specific gravity of microtome sections of tissue. The one finally adopted as most satisfactory consisted of dropping the section of tissue into one of a series of tubes each of which contained a liquid having a greater specific gravity than that of the preceding tube, and observing whether the section rose or fell in the liquid. If the section rose the next section was cut and placed in a tube of less dense liquid; if it fell it was placed in a tube of more dense liquid. In this manner the tube in which the section remained suspended was found, and the specific gravity of the liquid in this tube was taken to be equal to the specific gravity of the section of tissue. The sections were so thin (30 microns) that the specific gravity did not change appreciably from one to the next, so that several consecutive sections could be

TABLE 2.—*Amount and Specific Gravity of Liquids Used*

Chloroform, cc.	0.7	0.8	0.9	1.0	1.1
Alcohol, cc.	1.0	1.0	1.0	1.0	1.0
Specific gravity	1.077	1.099	1.118	1.131	1.149

taken to determine the final tube. These sections were always taken adjacent to one that had been removed for titration, so that proper correlation could be made.

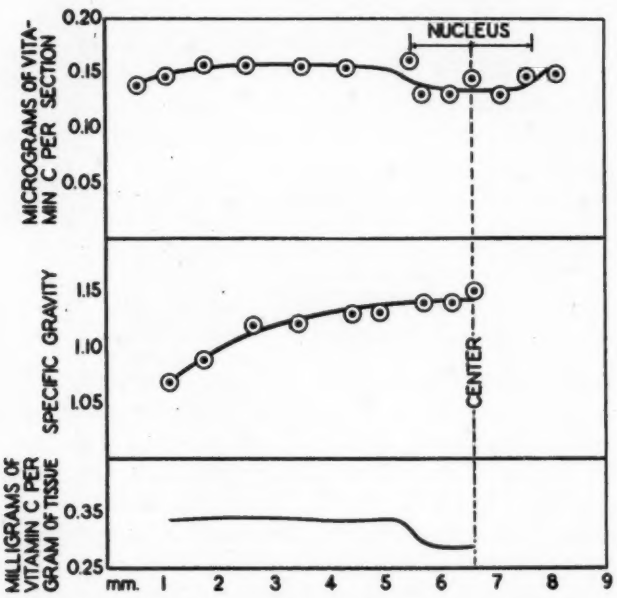
Five tubes were prepared by adding 1 cc. of purified petroleum benzine to each and 1.1 cc. of chloroform to the first tube, 1.2 cc. to the second and so on until 1.5 cc. was added to the fifth tube. The specific gravity of these mixtures increased regularly from 1.075 to 1.140. The mixtures of petroleum benzine and chloroform are desirable since they do not permit either removal or addition of liquid to the tissue once the sections are immersed. The lens contains no significant quantity of fat to be extracted, and the constituents of the tissue are insoluble in the organic medium. However, a difficulty was experienced with these mixtures. The gelatinous lens tissue could not be readily shaken off the glass needle used to carry the section from the microtome to the tube, and if the section should touch the glass wall of the tube after it had been immersed in the liquid it adhered firmly and was very difficult to shake loose.

The difficulties with the mixtures of petroleum benzine and chloroform resulted in the substitution of mixtures of alcohol and chloroform, which proved to be more practical. The latter have the disadvantage that the alcohol tends to dehydrate the tissue and coagulate the protein. But if observations are made within thirty seconds after the section is dropped into the liquid the effects of dehydration and coagulation are not sufficiently pronounced to influence the results of the measurements. The amount of the series of liquids finally used and their specific gravity are shown in table 2.

In the manner described, determinations of the concentration of vitamin C and of the specific gravity were carried out on four cow and three calf lenses. Each

lens was taken from a different animal. Because of the consistent results obtained it was considered unnecessary to employ more lenses.

In plotting the results curves were made by taking the micrograms of vitamin C per section and the specific gravity as ordinates and the distance in millimeters from the anterior surface of the lens as abscissas. From these data curves representing the milligrams of vitamin C per gram of tissue at different positions through the lens could be constructed. To determine the diameter of the nuclei of the lenses for these curves, lenses of the same size as those used for the study of the concentration of vitamin C were pressed carefully between the fingers to extrude the relatively firm nuclei, which were then measured with a vernier caliper. The borders of the nucleus are not sharply defined, but an approximation of the size of what is considered to be the nucleus can be obtained in this manner.



Curves for the concentration and the specific gravity of vitamin C in a cow lens.

COMMENT

The figure demonstrates the results of a typical experiment on a cow lens. It may be seen that the concentration of vitamin C is constant from the outer surface of the lens to the nucleus. In the nucleus a slight drop occurs.

The calf lenses showed practically the same curve for specific gravity. The other two curves were of the same shape but were from 10 to 15 per cent higher than those for the cow lens.

There are other reducing substances in the lens which it was thought might affect the dye used for the titration of vitamin C. Birch and

Dann [9] and von Euler and Malmberg [1b,d] attempted to check the titration method by feeding experiments, but apparently lens contained toxic substances which interfered with a quantitative correlation. They concluded that the vitamin C content is probably two-thirds the amount suggested by the dye titration method. Demole and Müller,[10] also using feeding methods of assay, reported that the antiscorbutic effect of lens is equivalent to one-half that indicated by the dye titration method. However, a recent paper by Rosner and Bellows [11] indicated that the reduction of the dye by the lens is entirely due to the vitamin C. This was demonstrated by the fact that the dye-reducing capacity of lens tissue is completely lost if the lens tissue is placed in contact with an enzyme that specifically oxidizes vitamin C (cevitamic acid oxidase).

SUBSEQUENT NOTE.—After this manuscript was submitted for publication a report by Johnson [12] appeared comparing the indophenol titration method of determining the vitamin C content of the lens with the spectrophotometric method. He found that the indophenol titration method invariably gave results which were higher than those obtained by the spectrographic method.

SUMMARY

The specific gravity and, by means of the 2, 6 dichlorophenolindophenol dye, the concentrations of vitamin C in various regions of the lens from the periphery to the center have been investigated.

A slight decrease in the concentration of vitamin C in the nucleus was observed when compared with that of the rest of the lens.

9. Birch, T. W., and Dann, W. J.: Ascorbic Acid in the Eye-Lens and Aqueous Humor of the Ox, Biochem. J. **28**:638, 1934.

10. Demole, V., and Müller, H. K.: Ueber das Vorkommen von Ascorbinsäure in Linse und Kammerwasser, Biochem. Ztschr. **281**:80, 1935.

11. Rosner, L., and Bellows, J.: Ascorbic Acid Oxidase in Determining Vitamin C in Lens and Aqueous Humor, Proc. Soc. Exper. Biol. & Med. **34**:493 (May) 1936.

12. Johnson, S. W.: CCIII. Cataract and Ascorbic Acid in the Guinea-Pig Eye, Biochem. J. **30**:1430, 1936.

OPERATIVE TREATMENT OF DETACHED RETINA

PRINCIPLES OBSERVED BY SIX INDIVIDUAL OPERATORS

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In a paper on general surgical subjects which I presented before the Brooklyn Ophthalmological Society on April 16, 1936, I read six résumés which I had previously obtained from the surgeons who will be named.

Each résumé was the personal expression of the points which seemed to the wirter to be most important in operating for detachment of the retina. It is seldom that one obtains an expression of opinion of this nature, and these citations of ophthalmologists distinguished and active in this work seem too valuable to remain unpublished.

As the communications were personal, I have obtained the consent of the author in each instance, and quote them in alphabetical order.

Mr. James Cole-Marshall, of London, wrote:

As regards the principal points in operating for detachment of the retina, I think that the operator should first make a careful examination of the patient and a thorough investigation of the eye, making drawings and persevering until a tear is found, I feel that it is to be found practically always. Then, of course, the next important thing is marking out the tear. I feel that Gonin's method is still as good as any other, except that it now can be supplemented either by using Vogt's cathode method (putting in a thin needle and seeing the bubbles which are given off at the point where the needle entered the vitreous) or by using a fine diathermy needle and obtaining an area of coagulation on the retina.

Another good point is using a reflected light in a dark room, which is conducted through the pupil to the hole. This area of light is seen on the sclerotic This method is used by Arruga and Šafář. For detachments which are very far back Manin's perioscope can be used.

But the main concern is to aim at closing the tear, whatever method is used.

I think that one should bring out the importance of draining off as much fluid as possible.

I am rather nervous about discussing these principles, as there are such distinguished authorities in America—Knapp; Clifford Walker; Walsh, of Johns Hopkins University, Peter and others. On the other hand, I do feel that these points are very important.

The tendency lately, with the new methods, is not to take trouble in finding the tear and marking it and not to aim at closing it. Weve is very emphatic on this point.

Dr. Arnold Knapp, of New York, expressed the following views:

The most important feature is the examination of the patient. For this the pupil has to be fully dilated. The patient must be repeatedly examined until the detachment has been carefully studied and the hole or tears located. When these have been located the meridian or distance from the limbus must be carefully and accurately determined.

Sometimes it is necessary to have the patient wear stenopeic glasses and remain flat on his back for a number of days, during which repeated examinations are made, before the nature of the detachment is fully understood. Then the operation may be performed with the patient under local anesthesia, a deep injection being made, with canthotomy and temporary tenotomy of one of the muscles if necessary.

The scleral region which corresponds to the affected retinal tissue is exposed, and the diseased area of the retina is then delimited by a series of surface coagulations, proceeding from the ora serrata and returning to the ora serrata, if possible, enclosing the affected area. The surface coagulations are best applied by means of the pyrometric method of Coppez, by which the degree of temperature to which the tissues are heated can be read directly on a scale. The endeavor is to produce slowly a temperature of 80 C. during an interval of twenty seconds. After the area has been outlined by the surface coagulations, a number of punctures with a very fine needle are done to let off the subretinal fluid. At the end of the operation the retina must be in position, as determined with the ophthalmoscope; otherwise the operation has not accomplished its purpose.

The accuracy of the localization must be controlled by observations with the ophthalmoscope.

The patient is kept perfectly quiet on his back for two weeks, with the head placed so that the area of the detachment is most dependent.

Dr. Luther C. Peter, of Philadelphia, wrote:

The important phases of the modern methods of handling retinal detachments, in my experience, are the following:

1. Early operation, i. e., within three months after the detachment.
2. Careful localization of tears.
3. Surrounding all the retinal tears by points of coagulation, the needles being placed about 2 or 3 mm. apart.
4. In cases in which no tears are visible, placing of needle points about 3 mm. posterior to the ora serrata throughout the detached area, accompanied by points of surface coagulation so as to include all areas of retinal elevation.
5. Free drainage (*a*) through needle points of coagulation, (*b*) through an opening made by a single incision and (*c*) occasionally through a trephine opening at the peak of the detached area.
6. Detachment of a sufficient number of muscles so as to give proper exposure.
7. In making the prognosis, careful distinction between the reattachment of the peripheral part of the retina and the restoration of central vision.

In my experience, if the tear is close to the macula or if macular vision has been disturbed for more than three months by simple elevation or edema, central vision will not be perfectly restored. Useful vision, however, usually follows, and after reattachment of the retina the complication of complicated cataract, as a rule, does not occur. I have operated on many patients with detachment of more than a year's standing, with excellent results so far as the reattachment of the retina is concerned, but in such cases macular vision is rarely restored to full function.

The cases in which the problem is most difficult are those in which detachment follows extraction of a cataract, especially of a congenital cataract which has been treated by repeated needling. In high myopia also the prognosis is not so favorable. It is fair, however, to estimate that the detachment can be arrested and the retina can be reattached in at least 85 per cent of all the cases. Central vision is influenced by the proximity of the tear to the macula and by the length of time that macular vision has been disturbed as a result of the detachment.

Dr. Mark J. Schoenberg, of New York, wrote as follows:

In my experience, taking ample time in recording all the details of the personal history of the patient and the history of the beginning of the trouble may furnish information not obtainable in any other way.

The second important point is the proper and thorough examination of the eye, especially of the ocular tension, media and fundus. The complete dilatation of the pupil is frequently neglected by a number of ophthalmologists; on this hinges sometimes the discovery of one or several tears which may be very near the ora serrata.

Then a complete record, including a drawing of the changes of the fundus, and their location, size and appearance, is absolutely essential. This record must be not only on paper but in the memory center of the surgeon so that he can visualize the fundus during the operation.

Following this step is the proper performance of the operation, which implies thorough mastering of the various steps and great care of handling the tissues with the utmost delicacy and tenderness. Apropos of the technic, one can speak of it only in general terms, since it would take too long to mention the numerous details of the various methods employed at the present time. Perhaps it may be of benefit to give a list of "Don'ts," which I have gathered from my own experience:

1. Do not use instillations of cocaine. This injures the corneal epithelium and interferes with ophthalmoscopic observation during the operation.

2. Do not allow the cornea to dry up during the operation, for a reason identical to that mentioned under section 1.

3. Do not lose sight of the area to be treated while the eye is pulled in the desired direction and the vertical axis is rotated.

4. Do not cauterize or injure the ciliary region, the long ciliary arteries and nerves, the venae vorticosae, the optic nerve or the nerves and blood vessels in its vicinity.

5. Do not puncture deeper than the choroid.

6. In using the diathermic current, do not injure the lids, especially the margin of the lids.

7. Do not use the diathermic current on a moist surface.

8. Use only the minimum intensity of current that produces the necessary effect.

9. Do not forget a sponge in the wound.

The after-treatment, which has been so thoroughly standardized that there is almost no divergence of opinion regarding it, consists of (*a*) absolute immobility of the patient and (*b*) complete mental and physical rest. The late after-treatment, which consists of the wearing of stenopeic lenses and avoiding undue strain of the body and eyes for at least two months following the operation, is as important as the other points just enumerated.

Dr. John M. Wheeler, of New York, expressed the following opinions:

My associates and I have developed ideas a little different from those of some ophthalmic surgeons regarding the operation for retinal detachment and the prognosis.

1. In regard to the intra-ocular tension, we consider the tension very important in the prognosis. Operation is almost sure to fail in cases of hypotony. Tension within normal ranges is one favorable prognostic point. Hypertension, which occurs occasionally with retinal detachment, is not a bad prognostic sign. Usually the tension is reduced by the operation for detachment.

2. My associates and I do not limit the treatment to the areas around the retinal holes but treat the entire area of the detachment. We are inclined to give special attention to areas around retinal holes, but perhaps even this is not important. We are now using the Lacarrère diathermy attachment that was designed for extraction of the lens, but we use a very fine wire singly instead of doubly, as Lacarrère and Arruga have done. We make the fine punctures in the sclera about 4 mm. apart. In cases of complete detachment of the retina we treat one hemisphere, and then, after a few weeks, if the eye has regained its tone we treat the other hemisphere.

3. Severity of treatment. Probably it is not necessary for most of the punctures with the fine wire to penetrate the subretinal space, and my associates and I aim to produce as little traumatism as feasible. We have no rule to guide us, but we have been inclined to err on the side of conservatism. Probably every puncture should penetrate at least to the choroid. Many punctures with frank choroidal reaction over the entire area concerned seem proper.

Dr. Clifford B. Walker, of Los Angeles, wrote as follows:

I reserve the word "microneedle" for the description of galvanic needles only, and always refer to micropins in association with diathermy, since that association has become so well fixed from the work of Šafář.

It seems to me that the use of the galvanic current, with perhaps a more refined technic than that originally described by Vogt, is such a distinctly valuable recent addition to the treatment of separated retina that I never treat a patient without having both negative and positive galvanic current, as well as diathermic current, instantly available This is described in my second article in the 1935 "Transactions of the American Ophthalmological Society."

The galvanic current is a valuable accessory, even if used only for localization. It will cure the condition in many cases, exactly as Vogt has described, but it will not cure the condition in all cases—a point that he has not emphasized much. I think that if the proper balance of the amount of galvanic current is used the amount of diathermy necessary and thereby the traumatism to the eye will be reduced, and a higher quality of vision will result in perhaps 90 per cent or more of the ordinary run of cases.

I have had correspondence with Mr. Cole-Marshall, of London, and with Colonel Robert E. Wright, of Madras, India, both of whom have tried some of my devices. I feel sure that my attitude toward the use of the cathodal current will not have to be changed much. I believe this to be the best operating room method of localization for flat and moderately elevated tears, and rarely is a torn area of retina so turned into the radius of the eyeball along which a galvanic needle must pass that it cannot be satisfactorily struck in all the anterior portions

of the eye. Even in such cases it is usually possible to strike margins of the hole with a curved needle or when drainage of the eye has changed the plane in which the tear lies with regard to the sclera.

I do not at present believe that any form of surface treatment—thermocouple controlled, bipolar or chemical—can possibly meet the demands in some of the conditions that are controllable with a combination of catholysis with the use of microneedles and efficiently reduced diathermy with the use of micropins and, occasionally, by undermining the Lindner treatment with potassium hydroxide. Treatment of macular holes by means of cathodal current and tears in the region of the posterior pole is the easiest, safest, quickest and best of all methods, with the use of either my right angle tip automatic microneedle or the plain right angle tip (2 or 3 mm.) iridium-platinum microneedle.

Reduction in the number of sutures and great reduction in the frequency of temporary tenotomies as well as in the amount of sclera exposed are necessary with a view to the reduction of traumatism. Such reduction comes with experience and accuracy of measurement and with the substitution of muscle splitting but is limited by the risk of another operation in cases in which under-treatment results. Strict asepsis and excellent anesthesia are absolutely necessary, and the use of every trick to maintain a perfectly clear cornea throughout the operation is of enormous advantage. Generally speaking, I feel that the preliminary localization and the planning of the operative procedure should be carried out during a half-day or more when no other patients are seen and no disturbance is possible.

In considering the sharpshooting method of ignipuncture in the use of galvanism as regards accuracy one is reminded of the original attitude of Gonin. One may now use dozens of galvanic microneedle shots without penalty for stray shots, whereas Gonin was practically limited to one shot at the tear with the method of ignipuncture. These subjects are all fairly easy to discuss, but I have never had to carry out anything more difficult, requiring more study of all the millimeter topography and variations of the inner and outer ocular coats. To obtain a certain percentage of cures is not difficult, but to obtain reattachment percentages above 90 in more than 90 per cent of a large group of cases of ordinary separations requires much study and forestalling, with accurate drawings of details in the region of the tear that will facilitate rapid ophthalmic observation.

The treatment of macular hole or degeneration seems to be necessary in one case of from eight to twelve. The macula should be invariably closely studied for the purpose of detecting cystic or some degenerative disease. I believe that it will have to be treated still more frequently than in 12.5 per cent of the cases to maintain the highest percentage of success. At present I reserve Lindner's undermining operation for court of last resort, perhaps the third operation.

A weak ophthalmoscopic light is a menace, and the best ophthalmoscope is none too good. I use the Friedenwald ophthalmoscope constantly, and for operative purposes I use a sterilizable ophthalmoscope that I have devised myself from an old instrument. This gives a sharp, square, penetrating beam without diffusion, when it is provided with a fresh bulb and batteries for each operation.

BIOCHEMISTRY OF THE LENS

X. PREPARATION OF GLUTATHIONE FROM THE CRYSTALLINE LENS

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AND
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The energy arising in the animal body is mainly the result of oxidative processes. Oxidation in the tissues is carried by two mechanisms: the addition of oxygen to a substance and the withdrawal of hydrogen from it. According to Duke-Elder,[1] the oxidative activity of crystalline lens is greater than that of nerve but less than that of muscle, while the oxygen tension in the aqueous is insufficient to meet the needs of the lens. According to Goldschmidt,[2] the lens is dependent largely on the second method of oxidation (the removal of hydrogen from the lens).

This mechanism depends on the presence of a substance that can act as a hydrogen acceptor. There are at least two such substances present in the lens (glutathione and vitamin C). The facts that glutathione is in greater concentration in this tissue than in any other and that this substance is diminished or lost in the cataractous lens lend weight to its importance in the oxidative process of the lens. The presence of glutathione in the lens is demonstrated by the use of a color reaction which it gives with sodium nitroprusside, which responds to sulfhydryl compounds. It is absent in the cataractous lens. Glutathione is readily oxidized if exposed to the air at body reaction. In the normal tissues, however, it is present almost exclusively in the reduced form. The reason for this is that the tissues contain a substance which reduces glutathione. This substance is called the thermostable residue because it is active even after it has been washed, boiled, extracted with alcohol and finally dried and powdered. In the lens the thermostable residue is betacrystallin (Adams [3]).

In addition to its respiratory function, glutathione seems capable of detoxifying certain substances. Bourne and Young [4] have demonstrated that rabbits fed naphthalene excrete a derivative of mercapturic

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1. Duke-Elder, W. Stewart: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, p. 479.
2. Goldschmidt, M.: Arch. f. Ophth. **113**:160, 1924.
3. Adams, D. R.: Proc. Soc. Med. **98**:244, 1925.
4. Bourne, M. C., and Young, L.: Biochem. J. **28**:1803, 1934.

acid formed by the conjugation of naphthalene with cysteine. One of us (J. B.) [5] has shown that the glutathione content of the lens diminishes before the cataract appears in rats fed a high galactose diet. If the sulfhydryl content of the diet is increased, the time of onset of the cataract is delayed. Further work along this line is contemplated. From these experiments one may assume that glutathione (or its cysteine portion) takes part in detoxifying certain substances. If the amount of the sulfhydryls (glutathione and cysteine) removed from the lens for the purpose of detoxication is great enough, a point will be reached where the oxidative function is insufficient to maintain the life of the lens. As a result of this a cataract forms.

Glutathione was first prepared from yeast and liver by Hopkins in 1921. Pirie [6] attempted to prepare it from muscle and blood but obtained unsatisfactory results. Up to the present time glutathione has not been isolated in pure form from the lens, according to Krause.[7] At his suggestion we undertook to obtain it from this tissue. Previous

Determined Values for Glutathione as Compared with Theoretical Values

	Determined Values	Theoretical Values
Melting point	183° (uncorrected)	190°
0.5 mg. of substance titrated with N/1,000 potassium iodide	1.64 cc.	1.63 cc.
Nitrogen (method of Dumas)	12.88 %	13.08 %
Sulfur (method of Pregl)	10.16 %	10.45 %

estimations of the amount of glutathione in the lens have depended on tests of reducing properties which are common to glutathione and several other substances.

EXPERIMENT

The mixture used to extract glutathione from the lens was that described by Pirie. It is a mixture of alcohol, ether and sulfuric acid. The lenses of two hundred beef eyes were ground in a mortar with sand and 200 cc. of the extraction mixture. A pasty mass was obtained which was filtered by pressure on a Buchner funnel. The pressure was brought about by placing a thin sheet of rubber over the top of the funnel, which was made air tight. The filtrate was then treated with a suspension of cupric oxide to precipitate the copper salt of glutathione. After thorough washing by centrifugation, the copper salt was reduced with hydrogen sulfide. The solution was then filtered, and the filtrate was concentrated in vacuo to a few cubic centimeters. This was placed in the refrigerator over night to allow the glutathione to crystallize out. Crystallization was aided by seeding. The yield was 0.2 Gm.

The table shows the properties of the substance as compared with the theoretical values for glutathione. A deviation from the theoretical

5. Bellows, J.: Biochemistry of the Lens: IX. Influence of Vitamin C and Sulfhydryls on the Production of Galactose Cataract, Arch. Ophth., to be published.

6. Pirie, N. W.: Biochem. J. **24**:51, 1930.

7. Krause, A. C.: Personal communication to the authors.

values is explained by the fact that no recrystallization was attempted, owing to the small quantity of the product obtained. From these data one may conclude that the substance prepared by us from the crystalline lens is glutathione.

FORMATION OF NEW VESSELS IN THE VITREOUS

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In 1859 Coccius first described new-formed vessels in the fundus, and several years later Mauthner and Jäger (1869) reported several cases. The latter author was acclaimed as having given a masterly description of the appearance of this condition. Leber also furnished an exact explanation.[1]

In some unusual cases these formations grow into the vitreous with one end attached to the retina or the disk and the other moving freely. Frequently the condition is preceded by retinitis haemorrhagica or retinitis exsudativa, due to various causes. The hemorrhage may be so dense at first that the fundus is hard to see, and one must wait until the blood is absorbed before the new-formed vessels can be seen clearly. In certain cases, however, when hemorrhage is not dense, the proliferation of the vessels can be observed in the initial state. Exudate in the fundus, which appears in acute or chronic inflammatory processes, is a mass of cells, pigment granules or filaments, and if it is not resorbed it becomes organized into a membrane, cords or even large masses of connective tissue, which are penetrated by the new formations arising from the retinal vessels and growing into the vitreous. Such an exudate in the vitreous originates from inflammation of the surrounding structures.[2]

New-formed vessels may be connected with retinitis proliferans or may occur in cases in which retinitis, particularly the syphilitic form, has lasted a long time. In this case the vessels grow from the retina into the vitreous in the form of slender coils, which often rapidly become convoluted. But in a number of cases it is probable that these masses of connective tissue have been preceded by hemorrhages, which were poured from the retina into the vitreous and afterward became organized.

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1. Leber, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, W. Engelmann, 1898-1900, vol. 7, p. 127.

2. Fuchs, Ernst: Lehrbuch der Augenheilkunde, Leipzig, Franz Deuticke, 1903; Textbook of Ophthalmology, authorized translation by A. Duane from the 12th German edition, Philadelphia, J. B. Lippincott Company, 1911.

The most common cause of new vessels of this type is the spontaneous hemorrhage appearing in the retina and vitreous in young persons, particularly as a result of tuberculosis.[3]

In cases of arteriosclerosis, in which vessels are straitened and completely obliterated, new-formed vessels take the function of the preexisting vessels. Diabetes,[4] periphlebitis, arteritis and the encapsulated tumor are not infrequently the direct cause of the presence of these vessels.

New-formed vessels grow usually from the central arteries at or near the disk, and after a time they may cover the disk entirely. The condition sometimes appears as a single long vessel only, which may extend into the depth of the vitreous, sometimes in a very tortuous course. Not infrequently the vessels form a veil spreading over the fundus, through which the retina can be seen clearly. When moved by a sudden movement of the eye, they slowly settle to their former positions.[5]

When retinitis has subsided and the hemorrhage is absorbed, these new-formed vessels slowly retrogress and finally may even leave no mark or remain only as a light grayish strand.

Through the permission of Professor Meller, chief of the clinic with which I am associated, and with the cooperation of Dozent Urbanek, I am able to report in this paper several cases in which the condition was treated in this clinic.

REPORT OF CASES

CASE 1.—*History.*—H. L., aged 22, presented himself at the clinic in May 1927, with the complaint of contusion of the left eye, which he had received two weeks previously. He stated that from this time vision in the left eye had begun to fail. The family history was of no importance.

Examination of the Eyes: Examination of the left eye revealed photophobia and lacrimation, with slight tenderness on palpation. The lids, except for slight swelling, were normal. There was marked circumcorneal injection, with congestion of the conjunctiva. The cornea itself was somewhat cloudy and showed many precipitates on the posterior surface. The anterior chamber was deep and the aqueous turbid. The iris was swollen and dull, and its markings were indistinct, with dirty grayish coloration. The pupil was dilated, owing to instillation of atropine. The lens was free and the vitreous slightly cloudy. The fundus could be examined with difficulty, but the disk was distinctly evident and sharply outlined; the vessels were normal, and no macular changes could be detected. Vision was 6/9, with ability to read Jaeger's test type 1. The right eye was normal, and vision was 6/5, with ability to read Jaeger's test type 1.

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3. Meller, J.: Two Lectures on the Tuberculous Etiology of Uveitis, Vienna, Carl Ueberreuter, 1932.

4. Nettleship, E.: Chronic Retinitis with Formation of Blood-Vessels in the Vitreous in a Patient with Diabetes, Tr. Ophth. Soc. U. Kingdom **5**:159, 1888.

5. Flint, G., and Harrington, D.: New Vessel Formation in the Vitreous, Brit. J. Ophth. **18**:27 (Jan.) 1934. Harlan: Extensive Vascular Growth in the Vitreous, Tr. Am. Ophth. Soc., 1889, p. 426.

General Examination.—Adhesive pleurisy was shown on the anterior surface of the left lung. The examination of the heart revealed mitral stenosis and mitral insufficiency. The Wassermann reaction of the blood was strongly positive to repeated tests. Reactions to intradermal tests with old tuberculin and tubercle bacillus protein [6] were strongly positive.

Course of the Disease.—The patient was immediately admitted to the ward and was given antisyphilitic treatment (neoarsphenamine), followed by subcutaneous injections of tubercle bacillus protein twice a week, in addition to the usual local treatments. In spite of the energetic medication, the course of the disease in the left eye became progressively worse. There was hypopyon, with an increase in the amount of precipitates. The cornea become more steamy, with numerous dense grayish spots, which, however, did not occupy the whole area. A few days later deep vessels could be seen growing from the periphery of the cornea to the center. In other words, the cornea presented the appearance of interstitial keratitis! Iritis, however, remained the predominant feature of the disease. The vitreous became more cloudy, so that it was difficult to examine the fundus. Vision was 6/60, with ability to read Jaeger's test type 9.

Two months later the right eye became painful, with ciliary injection, cloudiness of the corneal epithelium, iritis and the formation of precipitates on the posterior surface of the cornea. In spite of all the precaution and treatment, the right eye progressed to a condition similar to that of the left. Vision in the right eye was 6/18, with ability to read Jaeger's test type 1. The strength of the tubercle bacillus protein at this time was increased to 0.0033 mg. Unfortunately, the patient insisted on going home and was not under observation until six months later, when he returned to the clinic. The condition of both eyes was more stationary. There were still a few large precipitates, and the cornea showed deep scars and obliterated vessels. In the iris were a number of small, sharply outlined nodules. The lenses were normal, and the vitreous in both eyes was still cloudy. Vision in the right eye was 6/18, with ability to read Jaeger's test type 2, and in the left, 6/24, with ability to read Jaeger's test type 3. Both eyes showed a large central scotoma for color, but the peripheral fields were normal.

Though careful examination revealed the vitreous to be cloudy, the right disk was hyperemic and slightly prominent, with a blurred edge. The veins were enlarged and the arteries normal. There were numerous hemorrhagic patches around the disk and along the course of the dilated veins. One of the hemorrhagic masses projected into the vitreous, and the free end swayed with the slightest movement of the eye. In the mass were numerous radiating, broad red streaks of new-formed vessels, the ends of which formed anastomoses (fig. 1).

At the temporal side of the disk another mass could be seen, with new-formed vessels arranged in the form of a veil, embedding the whole surface. The left eye presented a similar condition, but more regressive. The new-formed vessels could be seen in detail, some of them forming obliterated cords and others grayish strands, which branched as they projected forward. The Wassermann reaction of the blood was negative and that of the spinal fluid was positive. The reaction to subcutaneous injections of 0.01 mg. of tubercle bacillus protein was very strong. The patient was treated as usual, with progressive improvement of both eyes, and he was discharged from the clinic.

In August 1932 he returned for observation. The condition in both eyes was quiet, but there were still organized exudates on the posterior surface of the

6. The preparation used in these cases was tebeprotein, a tubercle bacillus protein described by Toenniessen (München. med. Wchnschr. **26**:957, 1926).

cornea. The capsules of both lenses showed old marks of synechiae. The fundus of the right eye was to be seen clearly, with practically no abnormal changes except the remains of new-formed vessels in grayish strands. The left eye showed nothing abnormal but whitish remains of hemorrhagic patches.

The following facts may be recapitulated: (1) The patient was comparatively young; (2) in spite of energetic antisyphilitic treatment, the keratitis in the left eye took its typical course; (3) the disease in the right eye also followed the typical course, in spite of all the precaution and treatment; (4) iridocyclitis remained the predominant feature of the condition in both eyes, and (5) the patient reacted very strongly to tubercle bacillus protein.

Fig. 1.—Drawing of the retina in the region of the disk of the right eye in case 1, showing new-formed blood vessels.

As one may learn from the literature and in the study of a number of cases, latent infection may flare up at a time when the resistance of the tissues is weakened by general or local disease.[7] This case is no exception; the patient believed that his vision began to fail after a trauma. Since the patient said he had not acquired syphilitic infection, it was assumed that the disease was congenital. The keratitis presented all the features of the interstitial form. It also must not be forgotten that the patient gave a strong reaction to tubercle bacillus protein,

7. Meller, J.: Tuberculosis and Its Relation to Spontaneous, Post-Traumatic and Sympathetic Ophthalmia, Tr. Ophth. Soc. U. Kingdom **54**:467, 1934.

which indicates allergy.[8] The vitreous showed marked dimness, which can be explained chiefly on the basis of the preexisting iridocyclitis. The etiology in this case could not be entirely determined, but as will be noted later, tuberculosis as well as syphilis can cause new formations of vessels of this type.

CASE 2.—*History.*—F., a man aged 55, presented himself at the clinic in March 1928, with the complaint of a foreign body under the eyelid of the left eye, which subsequently was removed. From this time, for three months prior to examination, he thought that his vision was failing.

The family history was of no importance.

Examination of the Eyes.—The left eye externally appeared normal, but scars were seen in the cornea. The vitreous was diffusely cloudy; the disk was indistinctly seen; two hemorrhagic patches occurred at its center, and at about 3 disk diameters on the temporal side there were also white streaks and patches. The lower portion of the retina was covered by a large hemorrhagic mass.

Vision: Fingers were counted at a distance of 1 meter. The right eye was normal, and vision was 6/6, with ability to read Jaeger's test type 1.

General Examination.—An active tuberculous process was shown in the lungs. Neither apex was clear. The Wassermann reaction was negative. The reaction to 0.0001 mg. of tubercle bacillus protein was positive.

Course of the Disease.—The patient was treated with tubercle bacillus protein. Seven months later the left eye remained in the same condition, and only the vitreous was clearer. Fingers were counted at 1 meter. The right eye was essentially normal. The patient returned to his home.

In October 1929 he again was admitted to the clinic because, as he stated, the right eye also had begun to fail. The right eye was externally normal. Examination of the fundus of this eye revealed that the vessels were straitened and the outline of the disk was blurred; the lower part was covered with hemorrhages, and hemorrhages occurred at the temporal side of the disk. At the nasal and the temporal side of the disk of the left eye were grayish-white strands, measuring about 1 disk diameter in width and 3 disk diameters in length.

Vision was as follows: With a correction of +2.50 sph. for the right eye fingers were counted at 6 meters, and Jaeger's test type 10 could be read. With the left eye fingers were counted at 3.5 meters. Cultures of the blood for tubercle bacilli (Löwenstein) were negative.

In December the retina of the left eye was completely detached, and there was no red reflex. Vision was limited to perception of movements of the hand.

The right eye was externally normal. There were diffuse hemorrhages in the retina, especially below the horizontal meridian. The area was completely covered by hemorrhagic masses, which projected into the vitreous. From the disk new-formed vessels ran forward into these masses in radial arrangement, the ends of which anastomosed.

Vision in the right eye was 6/18, with ability to read Jaeger's test type 2.

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8. (*a*) Nettleship, E.: Syphilitic Arteritis with Retinal Hemorrhage and Growth of New Blood-Vessels from the Disc into the Vitreous Humor, Tr. Ophth. Soc. U. Kingdom **4**:150, 1883-1884. (*b*) Urbanek, J.: Die Bedeutung der Tuberkulose für die entzündlichen Erkrankungen des Uvealtraktes, Berlin, S. Karger, 1929; Ocular Tuberculosis, Tr. Ophth. Soc. U. Kingdom **52**:227, 1932; (*c*) Die allergischen Erkrankungen des Auges, Wien. med. Wchnschr. **84**:707, 743, 772 and 801, 1934.

A month later the right eye presented slight ciliary injection, but the luster of the cornea was good. The anterior chamber was deep; the iris was slightly dull; the pupil was dilated medially with atropine, and posterior synechiae were presented in the nasal and lower portions. The lens was clear. The fundus remained the same. Vision was 6/12 with a correction of +4.00 sph., and Jaeger's test type 1 could be read. The left eye was externally normal, and the pupil reacted well to light. The retina was totally detached. Vision was limited to perception of movements of the hands. The dose of tubercle bacillus protein (subcutaneous injection) was increased to 0.05 mg.

In May 1930 the left eye was blind. Examination of the right eye revealed new-formed vessels which arose from the disk and projected into the vitreous and similar vessels in the lower portion of the retina (fig. 2). The old marks of hemorrhages could still be seen.

Fig. 2.—Drawing of the retina in the region of the disk of the right eye in case 2, showing new-formed blood vessels.

In July 1931 the patient returned to the clinic for observation. The left eye was blind, owing to detachment of the retina. The anterior segment of the right eye was essentially normal, and there were minute posterior synechial markings on the capsule. The vitreous was markedly cleared. Old traces of the hemorrhagic patches and new-formed vessels were still clearly evident, some in grayish strands.

It is obvious that this case was one of tuberculosis, which was secondary to the process in the lungs. In other words, the condition was tuberculous phlebitis of the retina, with resulting repeated hemorrhages into the vitreous.[9] Hemorrhage recurred so frequently that the

9. Theobald, S.: A Case of Recurrent Retinal Hemorrhage Followed by the Outgrowth of Numerous Blood-Vessels from the Optic Disc into the Vitreous Humour, Tr. Am. Ophth. Soc. **4**:542, 1885-1887.

vitreous of the left eye never cleared perfectly and ultimately the masses organized, with vascularization and detachment of the retina. The right eye was saved by the injection of tubercle bacillus protein, but it is difficult to be sure what rôle the treatment played. Evidently there was resorption, and the new-formed vessels, after having performed their function, began to retrogress.

CASE 3.—*History.*—S. W., a man aged 40, presented himself at the clinic in April 1932, with the complaint of seeing black spots, which at first resembled a veil and later became more dense.

The family history was of no importance.

Past History.—At the age of 12 years the patient had suffered from arthritis. He had trachoma at 13 and pneumonia once. He had always been weak in his earlier years, and the lymph glands of the neck could be palpated. Five weeks prior to the onset of the complaint for which he was admitted, he had recovered from influenza.

Examination of the Eyes.—The cornea of the left eye was not clear, especially at the position of from 7 to 8 o'clock. Numerous obliterated vessels extended from the peripheral portion to the center. Other parts of the anterior segment of the eye were essentially normal. Examination of the fundus revealed that the disk was normal, but that several hemorrhagic patches occurred on its nasal side, with white streaks extending into the surrounding area. Vision was 6/18, with ability to read Jaeger's test type 10.

The cornea of the right eye showed many obliterated blood vessels, but the eye was otherwise normal. Vision was 6/6, with ability to read Jaeger's test type 1.

General Examination.—Roentgenograms of the apexes of both lungs showed slight cloudiness. The teeth were in poor condition, owing to pyorrhea. The reactions were strongly positive to intradermal injections of tubercle bacillus protein in doses up to 0.00001 mg. The reactions to the Wassermann test and the complement-fixation test for the gonococcus were negative.

The patient was treated with injections of tubercle bacillus protein.

Past History.—At the age of 12 years the patient had suffered from arthritis. He had trachoma at 13 and pneumonia once. He had always been weak in his earlier years, and the lymph glands of the neck could be palpated. Five weeks prior to the onset of the complaint for which he was admitted, he had recovered from influenza.

Course of the Disease.—In March 1934 the patient returned to the clinic, with the complaint of failing vision in the left eye and black spots in front of the visual field of the right eye.

Examination of the Eyes: The anterior segment of the left eye was in the same condition as before, but the fundus could not be seen, since hemorrhagic masses occupied almost the entire area. Tests for vision revealed ability to count fingers at 3 meters and to read Jaeger's test type 4 with a correction of +5.00 sph.

There was ciliary injection in the right eye; the cornea was dull, with numerous precipitates on the posterior surface and obliterated vessels. The anterior chamber was deep; the aqueous was slightly turbid, and the iris was hyperemic, with dilatation of the vessels. The pupil was slightly displaced to the temporal side because of posterior synechiae at the position of from 5 to 7 o'clock. The vitreous was cloudy, and the disk with its neighboring parts, especially the

upper part of the temporal region of the fundus, was covered with hemorrhagic patches. Vision was 6/24, with ability to read Jaeger's test type 5 with a +5.00 sph.

The patient was admitted to the ward and was given injections of tubercle bacillus protein, in addition to the usual treatment. For several weeks the left eye remained in the same condition, while the inflammatory process in the right eye began to subside. The background, however, showed little change. Two months later the external inflammatory features of the right eye had completely disappeared, except for numerous old precipitates and posterior synechiae. The vitreous was still cloudy, but the fundus could be seen.

The disk was clearly outlined and slightly hyperemic; in the upper part of the temporal portion were hemorrhagic masses, and through these masses passed new-formed vessels, arising from the disk.

Fig. 3.—Drawing of the retina in the region of the disk of the right eye in case 3, showing radial arrangement of new-formed blood vessels.

These new-formed vessels were arranged radially like a spider-web and projected into the vitreous. When the eye moved they seemed to sway in the vitreous. There were also several small hemorrhages scattered about the vein in the upper part of the temporal region, which was dilated. The anterior segment of the left eye remained as before; the vitreous was still very cloudy, with large hemorrhages, and at the temporal side several whitish streaks extended to the periphery. The hemorrhages were apparently organized, although no minute detail could be described.

The patient was discharged, with instruction to come to the clinic for observation. From that time he came once a week for injections of liver extract and iodine preparations. The use of tubercle bacillus protein, however, was still continued.

In March 1935 repeated examinations of the patient revealed that the condition in both eyes was externally quiet. The vitreous of the right eye was clear,

although still a little cloudy; old marks of hemorrhage and the new-formed vessels in the upper part of the temporal portion of the fundus could still be seen clearly, extending like a fan from the disk into the vitreous. Some were thin and obliterated. It seemed that a retrogressive process was taking place in these vessels. The background of the left eye was also clear, but not to the same degree as that of the right eye.

At this time vision in the right eye was 6/18, with ability to read Jaeger's test type 2, and in the left eye, 6/24, with ability to read Jaeger's test type 3.

There is no doubt that this case was similar to the preceding one. The presence of obliterated vessels and opacities in the cornea was due to trachoma, which was successfully treated. The active process in the lungs and the scrofulous condition of the patient made him an easy prey to tuberculous involvement of other parts of the system, especially of tissues the resistance of which had been lowered by preexisting disease or trauma.

COMMENT

The formation of new vessels is of rather frequent occurrence. In inflammatory processes of the retina, especially of the disk, the tissues not infrequently show formation of small, mostly capillary, vessels which because of their delicacy often are not thrown into bold relief but merely increase the redness of the disk.

Ophthalmoscopically visible formations of new vessels also occur in cases of an intense inflammatory process of long duration in which there is circumscribed disturbance or cessation of the circulation or internal exudate.

I wish to present the histologic picture of the formation of new vessels in a case from the collection of Professor Meller. The eye was destroyed by secondary glaucoma in association with chronic tuberculous iridocyclitis (fig. 4). A regressive exudative membrane could be seen on the retina. The tender capillaries from the retina to the exudate were clear, while the rather shadowy exudative membrane could just be recognized.

Block (1904) demonstrated the interweaving of the inflammatory membrane by new-formed blood vessels.

Such formation of new vessels may also be due to preretinal formation of connective tissue, the so-called retinitis proliferans. Stimulated by recurring hemorrhages, combined ranks of striped and reticular connective tissue are formed in the retina and the membrane in the vitreous which often are also vascularized by outgrowths from these structures.

A remarkable formation of new vessels in the vitreous originating from retinal vessels occurs in some cases of vitreous and retinal hemorrhage or in hemorrhagic retinitis. In such cases the turbid vitreous,

which is slight in amount, is streaked with rather deep vessels, which form simple or complicated loops or are united in dainty nets. The hemorrhages into the vitreous may be considerable in the beginning, so that the fundus is covered. The development of vessels appears only after the blood is resorbed.

Usually the vessels originate on the disk, beside the central vessels, and in some cases, as it seems, in the retina.

If there is no new hemorrhage into the vitreous or the retinitis disappears, the vessels may slowly regress and at last disappear.

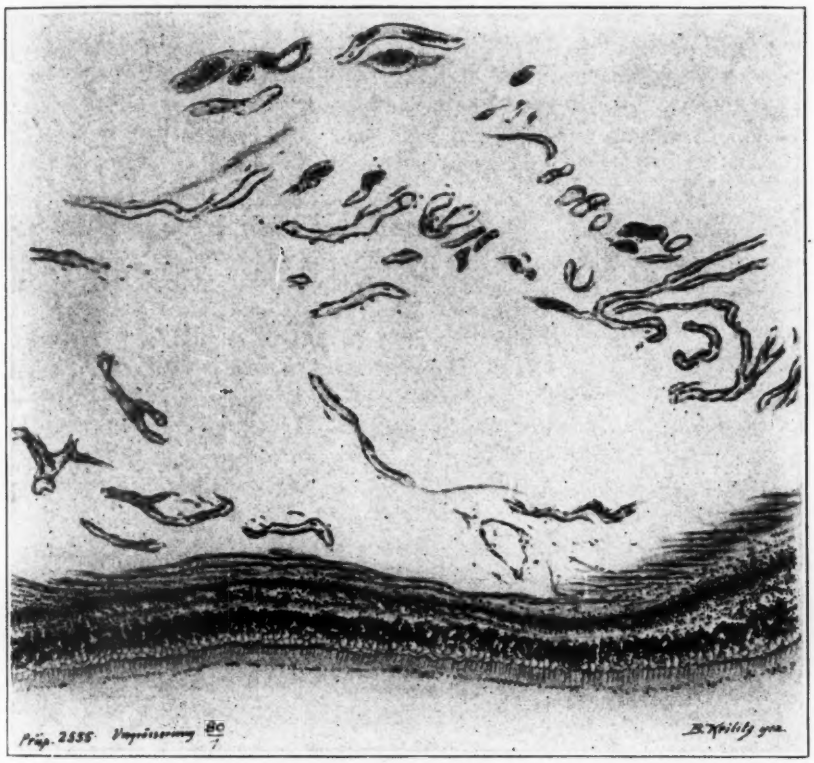

Fig. 4.—Drawing of a portion of the retina of an eye with secondary glaucoma in association with chronic tuberculous iridocyclitis, showing the formation of new vessels in a regressive exudative membrane.

The etiology of hemorrhage varies. In a number of cases the recurring hemorrhages of the vitreous and retina in young persons are connected with tuberculosis, as is shown in cases 1 and 3 in this series.

In the cases in which I made observations, disturbance in the vitreous, usually a hemorrhage, was always shown. Therefore, the moment of primary involvement of the retinal vessels was always known. Usually the condition was an inflammatory process of syphilitic or tuberculous nature. In most cases periphlebitis retinalis (Manz) was

observed, the etiology of which can be traced to tuberculosis. There are, however, without doubt cases of syphilitic involvement of the vessels.[8a] In these instances formation of new vessels due to arteriosclerosis could be observed, with high tension and thrombosis of the vena centralis retinae. In all the cases which we observed, until recently, periphlebitis and dimness (hemorrhage) of the vitrous were evident.

Therefore, in the cases in this series the newly formed vessels have the important function of resorption of the opacities of the vitreous and retina. Their mission having been fulfilled, the vessels always regress. This may be taken as proof that their function is only reparatory after the catastrophe of hemorrhage into the vitreous due to the tearing of retinal vessels. This explanation is offered for the existence of these vessels.

Clinical Notes

BILATERAL CYST OF THE VITREOUS

Report of a Case

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In the absence of detachment of the retina, nonparasitic cystic structures in the vitreous are extremely rare. They have been reported as varying in appearance from a free-floating semitransparent body to a cyst associated with or attached to a solid structure in the posterior segment of the eyeball. A thorough search of the literature reveals three examples of bilateral cyst of the vitreous.

The first case, reported by Vennin [1] in 1910, occurred in a highly myopic 22 year old soldier. The vitreous of each eye contained a floating greenish sphere, moving with movements of the eyeball and suspended from short filaments which appeared to fuse with the framework of the vitreous. The presence of these bodies produced no subjective disturbances or changes in the visual fields. The author believed them to be congenital remains of the hyaloid apparatus.

The second example, recorded by Lacarrere [2] in 1929, was in a patient with pigmentary degeneration of the retina. A flat cystlike structure was present on the nasal half of each optic disk. The author thought these bodies to be degenerative in origin and related to the retinitis pigmentosa, but he could not exclude a congenital etiology.

Litinsky's [3] case, presented in 1931, was in a 24 year old man with retinitis pigmentosa. The right eye contained a pea-sized round yellowish gray body floating in the vitreous, moving with movements of the eyeball and returning to the same location in the vitreous. The left eye contained a smaller but similar structure. The author saw fine fibers extending from the sphere to the lens capsule and to the ora serrata. He believed that the symmetry and round form of the cysts suggested an origin from the mesodermal elements of the embryonic vitreous.

The case to be described here was observed at the office of Dr. May and is reported with his permission.

Presented before the Section of Ophthalmology of the New York Academy of Medicine, March 16, 1936.

1. Vennin: Corps flottants bilatéraux et symétriques du vitré d'origine congénitale, Lyon méd. **119**:974-976, 1910.

2. Lacarrere, J. L.: Nota sobre un caso de formaciones quisticas hialoides, inflammatorias ? simetricas prepapilares, Conc. ophthal., 1929.

3. Litinsky, G. A.: Eine bilaterale runde symmetrische Bildung im Glaskörper, Klin. Monatsbl. f. Augenh. **87**:205- 208, 1931.

REPORT OF CASE

J. B., a 55 year old Russian tailor, gave a history of night blindness, restricted visual fields and failing vision for five years, with difficulty in distinguishing colors, especially blue and dark gray. There was no history of consanguinity or familial ocular disorder. The patient wore glasses to correct moderate myopia (—3.50 sphere).

Vision of the right eye was 15/60 with correction; vision of the left eye was limited to counting fingers at 1 foot (30 cm.). The visual fields were markedly

Figure 1

Figure 2

Fig. 1.—Appearance of the cyst in the vitreous of the left eye.

Fig. 2.—Appearance of the cyst in the vitreous of the right eye.

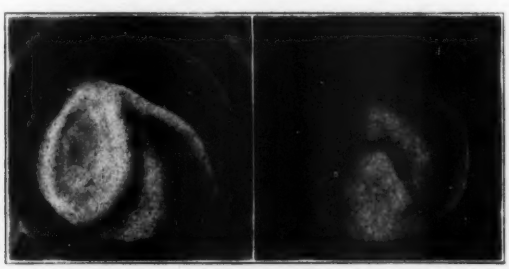

Fig. 3.—Cysts of the vitreous seen through dilated pupils: *A*, left eye; *B*, right eye.

constricted, and large ring scotomas were present. Both lenses contained posterior cortical opacities.

The fundi revealed pale disks, exceedingly narrow retinal arteries and multiple irregular pigment deposits in the peripheral portion of the retina.

Behind the left lens, floating freely in fluid vitreous, was a large yellowish gray globular translucent structure, measuring from 3 to 4 mm. in diameter (fig. 1). Its upper pole was elongated and somewhat indented. The surface of this cyst was smooth, glistening and avascular and contained no pigment. No strands connecting the body with other intra-ocular structures could be identified. On elevation of the eye, the cyst floated up or "bounced" into the optical and visual

axes, obstructing the fundus reflex viewed through the pupil. When the eye was motionless, the cyst sank to the dependent portion of the vitreous cavity.

The interior of the right eye revealed a similar but smaller cyst, which was somewhat oval (fig. 2).

The patient was aware of a momentary blurring of vision when elevating his eyes (fig. 3).

During six months of observation, there was no change in the appearance or size of the cysts.

COMMENT

The presence of a cystic structure in the vitreous unilaterally has been reported in twenty instances, but no one has had the opportunity of studying such a case in the pathologic laboratory.

The origins of such bodies have been more or less speculative, although in eight of the total twenty-four cases, the cyst appeared to be definitely related to a persisting congenital structure. The possible derivations of the cysts are as follows: The structure may arise from the fetal ocular cleft, mesodermal elements of the embryonic vitreous or the hyaloid apparatus. It may be a dislocated cyst of the ciliary processes, a formed exudate into the vitreous, a product of retinal degeneration (including retinal cysts) or proliferation or the result of degeneration of a parasitic cyst.

The presence of myopia or pigmentary degeneration of the retina or both of these conditions in the four cases of bilateral cyst suggests a congenital degenerative etiology.

70 East Sixty-Sixth Street.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

ACTION OF EPINEPHRINE ON THE DISEASED HUMAN EYE

USE OF THE STRONGER SOLUTIONS FOR INSTILLATION

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The value of epinephrine in the treatment of certain types of glaucoma is now well established. That complications may arise from its use is equally well known, and this knowledge creates the necessity of realizing the possibility of such ill effects and understanding them when they occur. Wessely [1] and Darier [2] were probably the first to demonstrate that the drug lowers intra-ocular pressure. These observations were corroborated both in the laboratory and in the clinic by other investigators.[3] Hamburger [4] has been the most ardent proponent of this form of therapy. It is due chiefly to his tireless efforts and extensive research that the method has become a recognized therapeutic procedure.

When first employed, epinephrine was injected subconjunctivally, usually in doses ranging from 0.3 to 0.5 cc. of a 1:1,000 solution. When it is realized that such doses represent a relatively large amount of active substance, it is not surprising that alarming reactions occasionally followed, especially in patients with vascular hypertension, cardiac disease, thyrotoxicosis, sympatheticotonia, and other disorders. A number of observers reported disturbing by-effects, including marked increase in blood pressure, palpitation, tachycardia (the rate exceeding 200), dyspnea and even collapse. This difficulty was circumvented to a large extent by topical application of the drug to the conjunctiva in the

1. Wessely, K.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **28**:69, 1900.

2. Darier, A.: Ocular Therapeutics According to the Most Recent Discoveries, translated by Sidney Stephenson, London, J. & A. Churchill, 1903.

3. Henderson, E. E., and Starling, E. H.: J. Physiol. **31**:305, 1904. Loewi, O.: Wien. klin. Wchnschr. **20**:1, 1907. Rubert, J.: Ztschr. f. Augenh. **21**:97, 1909. Erdmann, P.: ibid. **32**:216, 1914; Klin. Monatsbl. f. Augenh. **74**:413, 1925.

4. Hamburger, C.: (*a*) Med. Klin. **19**:1224, 1923; (*b*) Klin. Monatsbl. f. Augenh. **72**:47, 1924; (*c*) **76**:849, 1926; (*d*) Deutsche med. Wchnschr. **51**: 1397, 1925; (*e*) Ztschr. f. Augenh. **60**:109, 1926; (*f*) Treatment of Glaucoma with Glaucosan, Glaucosan Drops, and Amine Glaucosan Drops, Arch. Ophth. **55**: 533 (Nov.) 1926; (*g*) Brit. J. Ophth. **19**:455 (Aug.) 1935.

form of drops (Knapp [5]), on a pledget of cotton wool (Gradle [6]) or incorporated in an ointment (Thiel [7]). Occasionally the intra-ocular pressure was raised instead of lowered, causing an increase in the glaucomatous symptoms. According to Jess (cited by Hamburger [4g]), an acute rise in tension may be checked by the intravenous injection of 10 cc. of a 10 per cent solution of sodium chloride.

Hamburger then endeavored to devise preparations possessing the therapeutic efficiency of epinephrine but without its disagreeable side-effects. He first attempted to lessen the activity, thereby preventing reactions, by substituting the dextro-isomer of epinephrine.[4d] As will be recalled, epinephrine occurs in two optically rotatory forms, one rotating polarized light to the left (levorotatory) and the other rotating it to the right (dextrorotatory), a combination of the two types being known as racemic epinephrine. It has been definitely established that the active principle of racemic epinephrine lies in the levorotatory component, which possesses fifteen times the potency of the dextrorotatory form. Naturally, there is also a proportionate reduction in the toxicity of dextrorotatory epinephrine, and this probably influenced Hamburger to test the preparation clinically.

Epinephrine has been successfully synthesized by a number of independent workers.[8] When pyrocatechol (monohydroxyphenol) acts on chloracetyl chloride, the resulting compound is chloracetopyrocatechol. The latter when treated with methylamine forms methylamino-acetopyrocatechol, a product with a distinct pressor action. This substance can be converted into racemic epinephrine by reduction. The bitartrate salt is then formed, and by a process of fractional crystallization and purification it is possible to obtain the physiologically active levorotatory component (called suprarenin) absolutely free from the relatively inactive dextrorotatory isomer. Probably with the foregoing facts in mind, Hamburger combined one part of the slightly active dextrorotatory epinephrine with five parts of one of the ingredients used in its synthesis, methylamino-acetopyrocatechol, also having a pressor effect, and called the resulting preparation dextroglaucosan. Although dextroglaucosan was partially effective and free from unpleasant by-effects, it had to be administered by subconjunctival injection. Hamburger, recognizing the difficulties and disadvantages of having to inject the drug, sought to increase the potency so that it could be applied topically, by

5. Knapp, A.: Arch. Ophth. **50**:556 (Nov.) 1921.

6. Gradle, H. S.: (*a*) The Use of Epinephrine in Ocular Hypertension, J. A. M. A. **84**:675 (Feb. 28) 1925; (*b*) Illinois M. J. **53**:126 (Feb.) 1928.

7. Thiel: (*a*) Arch. f. Augenh. **96**:34, 1925; (*b*) Klin. Monatsbl. f. Augenh. **77**:753, 1926.

8. Funk, C.; Dubin, H. E., and Freedman, L.: J. Am. Pharm. A. **12**:952, 1923.

combining equal parts of synthetic levo-epinephrine with methylamino-acetopyrocatechol, naming the compound levoglaucosan. Levoglaucosan causes mydriasis and reduces the intra-ocular tension. The action often persists for several days. It is claimed to be effective in chronic simple glaucoma but less so in the acute form. From the foregoing discussion, it is obvious that the efficiency of both levoglaucosan and dextroglaucosan depends chiefly on the epinephrine content.

Hamburger then introduced a preparation which he called aminoglaucosan,[4c] a histamine-like substance (beta-imidazolethylamine) entirely unrelated to epinephrine. Aminoglaucosan stimulates smooth muscle, produces dilatation of the arterioles and capillaries and increases the permeability of the capillary walls, permitting the escape of plasma into the tissues. It causes a reduction in intra-ocular tension, and it is also considered the most powerful miotic known. The site of its miotic action is believed to be in the nervous system, since the pupil does not contract under anesthesia. However, since maximal miosis occurs with aminoglaucosan even with the eye under the influence of atropine, it seems more likely that the drug acts directly on the smooth muscle of the iris.

Aminoglaucosan, because its action is of short duration, was employed chiefly for the treatment of acute glaucoma. Although aminoglaucosan will promptly lower the intra-ocular tension, its application is attended by severe pain,[9] and consequently many ophthalmologists, after a short trial, abandoned the preparation. Furthermore, the results have not been uniformly successful. In a recent report, Hamburger [4g] confined his discussion to dextroglaucosan and levoglaucosan and made no mention of aminoglaucosan.

In the past few years attention has reverted to epinephrine,[10] which, there is reason to believe, will eventually replace the glaucosans in ophthalmology when the inherent value of each is finally determined. In a previous paper [11] I reviewed the literature on epinephrine and reported the results of a study of its action after topical application to the normal eyes of one hundred patients. It was shown that the instillation of a 2 per cent solution of a synthetic epinephrine preparation into the conjunctival sac of the normal eye is usually, though not always, followed by reduction in the intra-ocular tension. The fall in pressure sometimes

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9. (*a*) Duke-Elder, W. S., and Law, F. W.: Brit. M. J. **1**:590 (March 30) 1929. (*b*) Post, L. T.: Laevo-Glaukosan and Epinephrine Bitartrate in Treatment of Glaucoma, Arch. Ophth. **11**:187 (Jan.) 1934.

10. (*a*) Post.[9b] (*b*) Green, John: Two Per Cent Epinephrine Solutions as Substitutes for Laevo-Glaucosan, Arch. Ophth. **5**:350 (March) 1931. (*c*) Cordes, F. C., and Harrington, D. O.: Am. J. Ophth. **18**:451, 1935.

11. Howell, S. C.: Action of Epinephrine on Normal Human Eye: Use of Stronger Solutions for Instillation, Arch. Ophth. **12**:833 (Dec.) 1934.

occurred within one hour, but in most cases it was not observed until later. Concomitantly, the tension in the opposite eye in some cases was lowered, but to a lesser degree. The pupil dilated within an hour after application of the epinephrine and did not regain normal size until about twenty-four hours later. Maximal mydriasis persisted for approximately four to six hours. Beyond slight tachycardia and tremor, no untoward effects were noted, and even these symptoms may well have been due to the nervousness of the patient. It was concluded that epinephrine may be of aid in the treatment of certain types of glaucoma and may also be a suitable mydriatic for use in studies of the fundi and media of eyes in which the use of atropine or its derivatives is contraindicated.

Since the report of the preliminary investigation, ample opportunity has been afforded to study the usefulness of the method in diseased eyes. This can be illustrated by brief case records. In most instances a 2 per cent aqueous solution of synthetic epinephrine bitartrate was employed, although the drug may be incorporated in pure white petrolatum and used in ointment form. Some ophthalmologists prefer a 1 per cent solution,[12] the action of which is qualitatively the same, though quantitatively less, than that of the 2 per cent solution. For the preparation of solutions, special ampules of synthetic epinephrine bitartrate [13] were utilized. Each ampule contained sufficient epinephrine bitartrate powder to yield 0.05 Gm. of physiologically active levorotatory epinephrine base. To secure a 2 per cent solution, the contents of one ampule were dissolved in 2.5 cc. of sterile distilled water; for a 1 per cent solution the quantity of solvent was 5 cc.

A review of the voluminous literature on epinephrine and the glaucosans indicates that their greatest field of usefulness is probably in the treatment of glaucoma simplex. Sometimes when the tension in the glaucomatous eye cannot be maintained at a sufficiently low level with the customary miotics, this may be accomplished with epinephrine. In other instances the miotics become ineffective after a short period, and the tension rises to a dangerous degree in spite of vigorous use of physostigmine or pilocarpine. By changing to epinephrine one can generally arrest the progress and can cause the pressure to return more nearly to normal. When the application of the miotic preparations is resumed, it will be found that they have regained their former effectiveness. The tension occasionally can be controlled over long periods of time (a year or more) by epinephrine, used alone or in conjunction with physostigmine or pilocarpine. This is of particular importance in the case of old patients unsuitable for operation, in whom it is often possible through the use of epinephrine to avert blindness for their few remaining years.

12. Post.[9b] Cordes and Harrington.[10c]
13. The preparation used was suprarenin.

When operating for the removal of cataracts, some surgeons prefer epinephrine to atropine as a mydriatic for several reasons.[14] The principal advantage with epinephrine is that the pupil does not contract after the anterior chamber has been opened; this facilitates the surgical technic. Other desirable features are the lowered tension and the diminution in bleeding.

The value of epinephrine in glaucoma is shown in the following case reports.

CASE 1.—*Chronic simple glaucoma.*

A man aged 57 complained of gradually failing vision and frontal headache but had no pain in his eyes. The vision in the right eye was 6/28, and the left eye, 6/14. Manifest refraction improved the vision in the right eye to 6/14 and in the left to 6/12. The tension of the right eye was 36 mm. (Schiötz), and that of the left, 32. The pupils were large and unequal, the right measuring 5 mm. and the left 4 mm. The anterior chambers were shallow. Both visual fields showed upper nasal cuts in to within 5 degrees of fixation. Both disks showed cupping. Physostigmine was tried, but its use was discontinued because it caused severe pain in the eyes, nausea and vomiting. Pilocarpine was substituted, but the patient still complained of pain in the eyes and could not work. After four days of irregular treatment the tension was 32 mm. in each eye. Epinephrine bitartrate in 2 per cent strength was used in each eye every eight hours for three days. The tension was reduced to 22 mm. in each eye; the pupils were dilated, and the patient was comfortable. For the next eight months the patient was kept comfortable and without loss of vision or change in the fields by the use of one application of epinephrine every third day. At the end of this time a rise in tension occurred which could not be controlled with epinephrine. Iridectomy was performed on both eyes.

CASE 2.—*Chronic simple glaucoma.*

A man aged 67 complained of pain in the left eye, associated with loss of vision. The right eye was not affected. The tension of the right eye was 20 mm., but that of the left was 37 mm. (Schiötz). All other usual symptoms of glaucoma were present in the left eye. Four drops of a 2 per cent solution of epinephrine bitartrate was instilled into the affected eye at ten minute intervals. The next day the tension was 28 mm., and marked relief from pain was experienced. Treatment with a 0.5 per cent solution of pilocrapine hydrochloride three times daily was then instituted. Within two days the tension rose to 32 mm., with a recurrence of pain. By means of combined treatment with pilocarpine three times daily and epinephrine at weekly intervals, the tension was maintained at a fairly low level. An iridectomy was later done while the eye was entirely quiet.

In secondary glaucoma the results with epinephrine are in general not so satisfactory as in glaucoma simplex, but even in the former condition a most gratifying response may occasionally be obtained. The beneficial influence is shown by a reduction in the tension, with relief of pain, and by freeing of synechiae when these are present. The greatest improvement is observed in cases in which the pressure is

14. Cordes and Harrington.[10c] Barkan, O.: Am. J. Ophth. **15**:117, 1932. Beisgarth, C.: J. Missouri M. A. **32**:151, 1935.

not increased to a marked degree. However, in some instances, as in case 5, in which the tension was high, the reduction was startling.

CASE 3.—*Secondary glaucoma.*

A woman 40 years of age had pain in the left eye and complained of seeing halos around lights. The vision was 6/60. Examination showed the lids to be normal. The cornea was slightly bedewed, and there were fine deposits on the posterior surface of the cornea which were visible only with the slit lamp. The tension was 55 mm. (Schiötz). The visual field was not diminished when examined with a 10 mm. test object. The right eye was not affected. Atropine and hot applications were used at first. The tension ranged from 38 to 45 mm. for three days, with increase of pain. On the third day the tension was 45 mm. and the patient was uncomfortable. One drop of a 2 per cent solution of epinephrine bitartrate was instilled, and the patient became comfortable within an hour: The pathologist reported the Wassermann reaction of the blood to be 4 plus. Antisyphilitic treatment was immediately begun. By using epinephrine daily, or every other day, the tension was kept at a fairly low level as the inflammatory reaction subsided. Vision gradually improved so that at the end of two months it equaled that of the other eye, 6/5.

CASE 4.—*Secondary glaucoma.*

The patient was a man who had been treated elsewhere for secondary glaucoma three years before. When he was first seen he had moderate pain and some blurring of vision in the right eye. There was moderate iridocyclitis with some pericorneal injection. The pupil was fixed by complete posterior synechiae, but there was no iris bombé. The tension was 35 mm. (Schiötz). The left eye was not affected. After four instillations of a 2 per cent solution of epinephrine bitartrate the tension was reduced to 20 mm. The tension could be maintained at from 18 to 20 mm. by daily instillations of epinephrine, although the synechiae were not released. Two weeks after removal of two infected teeth the eye became quiet, and treatment was discontinued.

CASE 5.—*Secondary glaucoma.*

A woman aged 35 had repeated attacks of iritis during a period of fifteen years. Only the left eye was involved. All known foci of infection had been removed, but the attacks continued. On the occasion to be considered, she complained of more pain and greater blurring of vision than usual. The tension was 60 mm. (Schiötz). One drop of a 2 per cent solution of epinephrine bitartrate was instilled daily, and after three days the tension had fallen to 16 mm. The pressure remained low, and an uneventful recovery from this attack followed. Six months later the iritis recurred, without rise in tension.

For iritis, epinephrine is frequently a valuable agent because of its mydriatic action. Not infrequently, when atropine fails to release synechiae a strong solution of epinephrine is effective, particularly when used early. By combining the two drugs a maximal mydriatic effect is obtained, and this synergism may be utilized to advantage when either used alone proves unsatisfactory. Occasionally the addition of cocaine is of value.

In eleven cases of plastic iritis, posterior synechiae were freed by epinephrine after the energetic use of atropine had been ineffective.

A 2 per cent solution of epinephrine bitartrate was instilled four times at ten minute intervals. In seven of the cases all synechiae were broken up within one hour and in three within forty-eight hours; in the remaining case it required three instillations over a period of seventy-two hours before the iris was completely free. In four other cases of plastic iritis, neither atropine, used for twenty-four hours, nor repeated instillations of a 2 per cent solution of epinephrine bitartrate on three consecutive days proved successful. In two of these cases of failure, the eye ultimately quieted after six weeks of treatment, while in the other two cases the patient discontinued treatment after about two weeks.

The efficiency of epinephrine in iritis is shown by the following examples.

CASE 6.—*Iritis; posterior synechiae.*

A woman aged 52 complained of pain in her left eye, which had been diagnosed as iritis by a general practitioner, who treated her with atropine and hot applications. Examination on her first visit showed an inflamed, painful eye, with a partially dilated pupil and pericorneal injection. Fine opacities were seen on the posterior corneal surface, and two posterior synechiae were present. The anterior chamber was deep. Vision was 6/36 with correction. Atropine, cocaine and hot packs were prescribed, but no improvement had been obtained after twenty-four hours. Then four instillations of a 2 per cent solution of epinephrine bitartrate at ten minute intervals were made, and within one hour all synechiae were free and the pupil became fairly round. She made an uneventful recovery.

CASE 7.—*Iritis; posterior synechiae.*

A man aged 21 complained of burning pain in the right eye of ten days' duration. The pupil was bound down at about 2 mm. diameter. Atropine and cocaine had little effect on the posterior synechiae. The pain increased in the next two days. Epinephrine bitartarte, in a 2 per cent solution, was applied at ten minute intervals for four instillations. After the third instillation the synechiae began to pull away. Within one hour and ten minutes the pupil was round and free, 8 mm. in diameter, and the patient was comfortable. The Wassermann reaction of the blood was 4 plus. Antisyphilitic treatment was instituted, and an uneventful recovery followed.

When glaucoma is complicated by central cataract, miotics may not be desirable. Although they reduce the pressure, they constrict the pupil and further impair vision. Epinephrine, on the other hand, not only may reduce the tension but improves vision by reason of the mydriasis. This advantage is well illustrated in the following case.

CASE 8.—*Chronic simple glaucoma; central cataracts.*

A woman aged 70 had bilateral glaucoma of three years' duration, for which she had been treated in another city. She was made comfortable by means of physostigmine for about a year, but her vision gradually failed. One year prior to her first visit central cataracts developed while she was receiving treatment with physostigmine. Her vision was so reduced that she had to be led. Iridectomies somewhat improved her vision; but after two months the tension increased, and

on resumption of treatment with physostigmine her vision became so poor that she was again unable to "get about" without help. Examination of the right eye showed the cornea to be clear. The anterior chamber was shallow. A clean iridectomy had been done above. The pillars were free. There was a fairly dense central cataract. The coloboma of the iris (under the influence of physostigmine) measured about 2 by 6 mm. beginning 1 mm. below the center of the lens. The tension of the eye was 25 mm. (Schiötz). Vision consisted of ability to count fingers at a distance of 0.5 meter. The left eye was similar to the right except that the tension was 9 mm. and the vision was limited to light perception and projection in the lower nasal field only. One hour after instillation of a 2 per cent solution of epinephrine bitartrate the coloboma in each eye spread to about 4 by 6 mm. and the vision in the right eye improved to 5/60. The vision in the left eye was only slightly improved. By using epinephrine every other day the patient could see well enough to do her housework for several months, until she died of pneumonia.

In the following case of hemorrhagic glaucoma caused by thrombosis of the central vein no improvement was seen after the use of epinephrine. This, however, is not surprising, since all forms of therapy are generally ineffective against the apoplectic type.

CASE 9.—*Hemorrhagic glaucoma.*

A man aged 46 complained of aching pain and poor vision in the left eye. The vision was questionable light perception. The fundus showed thrombosis of the central vein, with characteristic massive sunburst hemorrhages. The tension was 35 mm. (Schiötz). The vision in the right eye was 6/6—2. There was marked sclerosis of the retinal arteries, and occasional small spots of exudate were noted throughout. Examination of the left fundus through the small pupil had not been satisfactory. After four instillations of a 2 per cent solution of epinephrine bitartrate into the left eye, the pupil enlarged to 8 mm. and a thorough study of the fundus could be made. The tension was 32 mm., and the patient was slightly more comfortable. Epinephrine was not used again. The pain continued despite persistent treatment with hot applications and opiates. In two days the tension rose to 82 mm. Iridectomy afforded relief for about two weeks, when the pain again recurred. The tension increased greatly, with much suffering, and enucleation was necessary.

The danger of local reaction from epinephrine applied topically is almost nil. When it was the custom to inject the drug subconjunctivally, a small superficial ulcer sometimes occurred at the site of the needle puncture, but it healed promptly without special treatment. The more powerful aminoglaucosan has been held responsible for a few reported cases of corneal irritation and hypopyon ulcer.[9a] In my experience with epinephrine I have observed only one instance in which there was any reason to believe that it may have caused a local reaction. A patient with old quiet iritis had a preparation of epinephrine instilled to release posterior synechiae and was allowed to go home on a windy day without a protecting eye bandage. On her return, after twenty-four hours, an excoriation of the anterior corneal epithelium was noted. It was thought

that this might have been due to diminished circulation and drying of the cornea. Such a complication may be easily prevented if the eye is closed and covered.

The application of epinephrine to the eye may be followed rarely by an initial rise in tension. Such rise in tension may continue for sufficient time and produce enough pain and pathologic change in the eye to necessitate surgical intervention. While such an occurrence is most unpleasant, I do not believe the small number of such cases can outweigh the great benefit obtained in many others. Descriptions of two such cases follow.

CASE 10.—*Chronic simple glaucoma; complication.*

An unmarried woman 60 years of age had pain in both eyes and marked loss of vision. The tension in each eye was 58 mm. (Schiötz). A 2 per cent solution of epinephrine bitartrate was instilled into both eyes, and a diagnosis of bilateral glaucoma simplex was made. The pressure diminished to 35 within two hours. A 1 per cent solution of pilocarpine hydrochloride was prescribed to be used three times daily. The patient went home and was comfortable until the fourth day, when she returned with recurrence of pain and loss of vision. Epinephrine bitartrate was again instilled. Within an hour she complained of extreme pain. Both eyes were stony hard to palpation and both corneas edematous. She was immediately hospitalized, and both eyes were trephined. She made an uncomplicated recovery.

CASE 11.—*Chronic simple glaucoma; complication.*

A man aged 64 had been a patient for refraction for the past five years. He had central choroiditis in the right eye and 6/60 vision with correction. The vision in the left eye was 6/6 with correction. About two years after the last refraction, he returned, complaining that the lids of the right eye seemed to "draw," that the vision was poorer than usual and that it was difficult to keep the eye open. The vision at this time was light perception only in the right eye and 6/6 in the left eye. The tension was 70 mm. (Schiötz) in the right eye and 20 mm. in the left eye. A 2 per cent solution of epinephrine bitartrate was instilled into both eyes. The fundi were examined, and the impression of glaucoma simplex in the right eye was confirmed. The left fundus was normal. Two days later the tension of the right eye was 25 mm., and that of the left eye was normal to palpation. A 0.5 per cent solution of pilocarpine hydrochloride was instilled into the right eye four times daily. Three weeks later he returned. The tension of the right eye was 45 mm., and that of the left, 22 mm. Epinephrine bitartrate was again instilled, and trephining of the right eye was advised. Three days later the tension of the right eye was 48 mm. Epinephrine bitartrate was again instilled. Two hours later he returned, complaining of great pain in the right eye. The tension was not taken with the instrument, but the eye was stony hard to palpation. Hospitalization, opiates and the usual regimen for treatment of acute glaucoma gave some relief from pain and reduction of tension to 40 mm. An Elliott trephination was done five days later without complication.

SUMMARY

Epinephrine bitartrate applied topically to the conjunctiva is a most helpful therapeutic agent for a number of diseases of the eye. Not

infrequently a condition may be successfully managed by its use when other customary measures are ineffective.

Many patients with chronic simple glaucoma, especially those for whom surgical intervention is inadvisable, are made comfortable by the regular application of epinephrine bitartrate, which reduces the intraocular tension to a more nearly normal level. When epinephrine is employed in conjunction with miotics, the action of the latter is often enhanced.

In cases of glaucoma complicated by central cataract, the vision may be greatly improved because of the mydriasis resulting from the instillation of epinephrine.

Since the pupil remains dilated even after the anterior chamber is opened, epinephrine is preferred by some ophthalmologists to atropine as a mydriatic for use in operations for cataract.

Epinephrine is a valuable mydriatic for examination of the fundus in glaucoma or other conditions in which a rise in pressure might prove dangerous.

Good results are sometimes observed in cases of secondary glaucoma, and the use of the drug should be considered when other drugs have failed to control the pressure.

Because of its strong mydriatic action, epinephrine is an efficient preparation for freeing synechiae in iritis.

Topical application of epinephrine produces fewer disagreeable by-effects than subconjunctival injection. The complication of sustained rise in tension following its instillation usually requires surgical treatment.

Drs. Grady Clay, William E. Campbell and J. Mason Baird permitted me to report some of the cases presented.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NEWS

Philadelphia County Medical Society.—The following program was presented at the scientific meeting of the ophthalmologic section of the Philadelphia County Medical Society, held on November 5: Dr. R. S. Minton, "Cases of Retrobulbar Neuritis Several Years After the Diagnosis Was Established," with presentation of patients; Dr. J. P. Guequierre, "Treatment of Ophthalmic Lues;" Dr. Robert Bookhammer, "Syphilis of the Central Nervous System from an Ophthalmological Standpoint;" Dr. Oscar V. Batson, "The Vascular Supply of the Central Nervous System."

National Society for the Prevention of Blindness.—The 1936 annual conference of the National Society for the Prevention of Blindness will be held from December 3 to 5 at the Deshler-Wallick Hotel, Columbus, Ohio. The following subjects will be discussed: "Saving Eyesight in Industry," "Eye Health in Relation to Social Work," "Rehabilitation versus Relief," "Conference on Sight-Saving Classes," "Eye Health of College Students," "The Nurses' Approach to Eye Health," "Teacher Education in Eye Health" and "Sight-Saving Classes in the Public Schools."

The annual meeting of the National Society for the Prevention of Blindness, Inc., will take place on Friday, December 11, at 4 p. m. at the Russell Sage Foundation Building, 130 East Twenty-Second Street, New York. A cordial invitation is extended to all members and friends of the society to attend this official annual meeting.

Correspondence

IRIDECTOMY WITH CYCLODIALYSIS

To the Editor:—Dr. Wheeler's paper read before the Section on Ophthalmology at the last annual session of the American Medical Association and published in the October issue of the ARCHIVES (**16:** 569, 1936) as well as the discussion following it ask for a comment.

The first to combine an iridectomy with a cyclodialysis was Czermak. His method was difficult—Czermak had a flair for highly complicated operations, for example, his subconjunctival extraction of cataract—and was published in 1906, the year of his untimely death. Elschnig modified Czermak's procedure.

Dr. Wheeler took pains to point out that his combined operation is a far more mutilating process than a Lagrange or an Elliot operation or an iridencleisis. I cannot well imagine this to be the consensus of ophthalmologists. The cyclodialysis, unless clumsily performed, is *the* nonmutilating operation for glaucoma; the only possible damage one can do is a peeling off of Descemet's membrane from the substantia propria of the cornea—a harmless occurrence the result of which compares favorably with the ugly filtering cushion above a hole in the sclera after trephination or with the unsightly scar after iridencleisis or iridotasis.

Dr. McReynolds, in discussing Dr. Wheeler's paper, stated that he prefers to make the scleral incision 2 mm. external to the limbus. I am unable to see how he can thus perform a detachment of the ciliary body sufficient to safeguard a proper drainage. Heine himself mentioned 5 mm. as the proper distance. Elschnig chose a point far more distant (from 8 to 10 mm.) in order to detach at least one third, or better two fifths, of the ciliary body.

Dr. Burch advocated an incision 3 mm. behind the limbus over a width of 8 or 10 mm.; his method, like that of Dr. McReynolds, chooses an approach too close to the limbus. Besides, the possibility or probability of the formation of a postoperative ciliary or intercalar staphyloma ought to be considered.

Dr. Woodruff's suggestion that the eye be massaged on the day after the operation deserves to be followed; it is a standard procedure in the peaceful treatment of simple glaucoma.

Dr. Gifford's remark that the cyclodialysis is the operation of choice for the troublesome glaucoma following the extraction of a cataract is well founded. Both Elschnig, in Czermak's "Augenaerztliche Operationen" (1908), and Heine, in von Graefe and Saemisch's "Handbuch der gesamten Augenheilkunde," failed to list this important indication for cyclodialysis.

I cannot agree with Dr. Gifford's skepticism about the lack of a permanent effect of the cyclodialysis. When properly chosen (tension not higher than 40 mm. Schiötz) and properly done with a sufficient arc of the ciliary body detached, it is effective in about 65 of 100 cases—not so bad a showing, after all, when one considers that one can repeat it ad libitum.

The only serious flaw in the cyclodialysis is the frequency with which it is followed by the formation of a cataract. However, this unhappy

occurrence is not rare in eyes which have undergone other antiglaucomatous operations, especially scleral trephination.

ERNST WALDSTEIN, M.D., New York.

ORIGIN OF OPHTHALMOLOGIC TERMS

To the Editor:—The following remarks may be of interest in rounding out the instructive article of Dr. Rodin in the October issue of the ARCHIVES (**16:** 609 [Oct.] 1936). The term "optist" has been used at one time or another by some makers of artificial eyes. It was printed on signs and must have been used in some publications, as it is now listed in at least one dictionary.

The term "opticist" was apparently used first by Dr. Swan M. Burnett in an article entitled "Why the Eyes of Animals Shine in the Dark" (*Pop. Sc. Monthly* **24:** 814 [April] 1884). The term was independently coined anew in 1886 by Mr. Charles F. Prentice, the father of optometry in the United States. Mr. Prentice, although he was the first president of the New York State Optometry Board and held the office for many years, disapproved the term "optometrist" and called himself an "opticist" and later also a "physical eye specialist." He wrote a little brochure relative to the term "opticist," which he copyrighted in 1886. Apparently neither he nor the copyright office knew of the appearance of the word "opticist" in the earlier publication. It is most likely that Dr. Burnett, who was a warm personal friend of Mr. Prentice and in a letter to him approved his use of the term "opticist," did not know that the term which he had originated had been copyrighted by Mr. Prentice.

The term "opthalmist," evidently a variation of "ophthalmist," has recently been adopted by a group of optometrists who formed an "Association of American Opthalmists" and apparently copyrighted the term, as it is stated in their announcement that no one may use the term who is not a member of their association.

I believe that the term "optometry" meaning "the measurement of visual power" was used in print before 1892. I remember distinctly having seen the term in a little book on "Diseases of the Eye and Refraction," published before 1892, but the little book has been lost or displaced; so I cannot check on it.

The term "optologist" has an amusing story associated with it. At a social gathering a gentleman was introduced to a professor of classical languages. In the course of conversation the gentleman told the professor that his occupation was that of optologist. The professor was intrigued by the term but did not know what it meant. However, he refrained from asking, figuring that he would find the word in the dictionary. On returning home he consulted the dictionary, but the word was not there. He then began to analyze the word and finally struck it, as he thought. "Op" is from the Greek for eye, "tolo" is from the Greek for potato; an optologist is therefore one engaged in raising eyeless potatoes.

Consulting the largest American dictionaries, one will find all the four terms—optist, opticist, optologist and ophthalmist—in "Funk and Wagnalls New Standard Dictionary of the English Language;" only three—opticist, optologist and ophthalmist—in "Webster's New International Dictionary of the English Language," and only two—opticist and ophthalmist—in the twelve volume Century Dictionary and Cyclopedia.

JOSEPH I. PASCAL, M.D., New York.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

POSTERIOR INSERTION OF THE ORBITAL MUSCLES. A. VILA-CORO, Klin. Monatsbl. f. Augenh. **96:** 466 (April) 1936.

Vila-Coro renders a detailed anatomic description of each of the extrinsic ocular muscles and of their arrangement around the optic nerve. He explains the condition of Zinn's ring, the function of which was ascribed by Zinn to the tendon of this ring. He arrives at the following conclusions: 1. The nasal and upper portion of the levator of the lid is covered by the superior oblique muscle. 2. The lower portion of the levator terminates in the temporal and upper portions of the medial rectus muscle. 3. The superior oblique muscle rests in the inner angle of the orbit on the upper portion of the medial rectus. 4. The superior rectus has a tendon which unites with the upper portion of the lateral rectus, producing a portion of Zinn's ring. 5. The lateral rectus is composed of two portions which connect with the superior rectus and the lateral rectus, respectively. 6. Zinn's ring is formed by two portions of the posterior insertion of the lateral rectus and the insertions of the adjoining muscles. 7. The inferior rectus fits into the lowest portion of the orbit, and its temporal portion helps to form Zinn's ring. 8. Three fibrous membranes are confluent in the lowest portion of the insertion of the inferior rectus: the periosteum of the inner walls of the orbit, that of the sphenoid cavity, and the dura mater which covers the middle fossa of the base of the skull. 9. The alleged tendon of Zinn does not exist.

K. L. STOLL.

Bacteriology and Serology

THE MECHANISM OF IMMUNITY IN OCULAR TUBERCULOSIS. E. KUNZ, Arch. f. Augenh. **109:** 706, 1936.

According to Kunz, agglutinins and complement-fixing antibodies play a part in the humoral defense of the body against tubercle bacilli. In order to investigate the formation of such antibodies, he injected killed bacilli into the anterior chamber of the eye in a series of rabbits. At varying intervals the complement fixation of the blood serum and of the aqueous was determined.

The experiments fall into two groups: In one group a single large dose was injected into the anterior chamber, resulting in a violent inflammatory exudation and keratitis; in the other group smaller repeated doses were injected in the hope of producing a less violent, more chronic type of inflammation.

In all cases there was a more or less marked reaction in the whole anterior segment of the globe. The iris became inflamed a few hours after the injection, and a serous-fibrinous exudate soon formed. In the eyes given massive doses the inflammation gradually increased in

violence, with increase in intra-ocular pressure and enlargement of the globe. In several cases phthisis set in after eight weeks. Histologic examination of these eyes showed a necrotizing chronic inflammation. In those eyes which received small doses of bacilli the inflammation gradually subsided.

No influence of the infection on the animals' general condition could be determined. Postmortem examination showed no changes in organs other than the eyes.

In the first group complement fixation in the blood serum was distinctly positive after eighteen days. It began to be positive after about nine days and remained faintly positive for as long as twenty weeks. The complement fixation in the aqueous, on the other hand, remained negative for more than eighteen days and became positive only in the course of the next fourteen days. In the second group complement fixation in the serum was only weakly positive throughout; that in the aqueous was negative in all cases.

Kunz concludes from these experiments that the formation of complement-fixing antibodies takes place outside the lesion containing the tubercle bacilli. The antibodies are produced in the blood stream by the antigen from the tuberculous lesion and get back to the site of the lesion only by transport through the blood stream. If the reticulo-endothelial system is chiefly concerned in the production of these antibodies, it is the entire reticulo-endothelial system of the body that is concerned and not just that adjacent to the lesion itself.

F. H. ADLER.

Biochemistry

A CORRECTION OF OUR PREVIOUS ARTICLE ON THE BLOOD-AQUEOUS BARRIER AND VITAMIN C. H. GOLDMANN and W. BUSCHKE, Arch. f. Augenh. **109:** 689, 1936.

In the previous article (*Arch. f. Augenh.* **109:** 205, 1935) the cevitamic (ascorbic) acid content of the aqueous was determined in a series of rabbits for each of the two eyes and the values expressed as percentages. The mean error of these percentages was given in the previous article as ± 2.02. This was a mistake, as the correct value is ± 6.4. This mistake did not in any way affect the authors' conclusions.

Since a comparison of the value of the cevitamic acid in the aqueous of the untreated eye serves for a control in many of their experiments, they have examined the aqueous of the two eyes in thirteen more normal rabbits and compared the cevitamic acid contents. They report again that the mean difference in the cevitamic acid contents of the two normal eyes is quite small and that in any experiments wherein one eye is treated experimentally the cevitamic acid content of the fellow affords sufficient control.

F. H. ADLER.

THE AMOUNT OF VITAMIN C IN THE AQUEOUS OF NORMAL AND OF PATHOLOGIC EYES. A. GALA and J. MĚLKA, Arch. f. Augenh. **109:** 726, 1936.

Gala and Mělka aspirated specimens of the aqueous from a series of patients and determined the cevitamic acid content for each. These

specimens were divided into those (1) from normal eyes without any sign of inflammation, (2) from inflamed eyes and (3) from eyes containing senile cataracts. In the first group the ages ran from 13 to 60 years. In the second group the ages were from 16 to 65 years, and in the third group the youngest was 55 years old and the oldest 80 years old. Altogether, 165 eyes were examined. Since both eyes were examined at the same time, a good control was afforded.

They found that normal eyes have, on the average, around 4 mg. of cevitamic acid per hundred cubic centimeters of aqueous, while eyes the subject of inflammation have around 3 mg. Of the seventy-four instances of cataract of one eye, in seven the cevitamic acid content of the cataractous eye was the same as that of the control eye; in sixteen it was less, and in eleven it was greater than that of the control eye. In four cases in which one eye was aphakic and the other cataractous the cevitamic acid content of the aphakic eye was higher twice and lower twice than that of the fellow eye containing a cataract.

The assumption that the cevitamic acid in cataractous eyes is less than normal is not justified as a cardinal rule.

F. H. ADLER.

Color Sense

HEREDITARY COLOR BLINDNESS: A GENEALOGY OF FIVE GENERATIONS. A. GROENOUW, Klin. Monatsbl. f. Augenh. **96:** 433 (April) 1936.

In 1920 Groenouw reported a genealogy of color blindness for red and green (Graefe and Saemisch: Handbuch der gesamten Augenheilkunde, ed. 3, Leipzig, W. Engelmann, 1920). He has now succeeded in completing this genealogy over five generations. A man and a woman of the first generation were not color blind. The disturbance originated most probably in the wife's family. A diagram of the genealogy is given in illustration. Among the thirty-two descendants there were eight color-blind men whose mothers were free from this disorder. Five women, including the one of the first generation, had color-blind sons, two had none, and the other women had no children. The color-blind men had no descendants, and two men with normal perception of color had no color-blind children. Color-blind women were absent, but eight of the nineteen men were color blind. Color blindness was absent in all the families of those who married into this family. The disturbance was transmitted according to the Horner-Bollinger type of heredity. The healthy women transmit the disorder to their sons but not to their daughters. Men free from color blindness did not transmit it to their descendants.

K. L. STOLL.

Conjunctiva

THE IMPORTANCE OF THE PHLYCTENULAS: REPORT OF CASES. R. G. POSTHUMUS, Klin. Monatsbl. f. Augenh. **96:** 362 (March) 1936.

Posthumus reviews the present state of the etiology of phlyctenulas, their relation to tuberculosis, rosacea and other diseases, and their affiliation with allergic conditions. He reports eight cases of phlyctenula associated with rosacea, in some of which it was also associated

with choroiditis or iridocyclitis. He points out the apparent connection of several phlyctenular types of conjunctivitis and keratitis with disturbances of endocrine secretion and associated manifestations of the autonomous nerve system. The résumé of his eight cases of phlyctenula associated with rosacea prompts him to suspect a tuberculous basis. He differentiates between genuine allergic conjunctivitides on the one side and scrofulosis and rosacea on the other. He thinks the two latter forms may represent an allergy against the antigen of tuberculosis.

K. L. Stoll.

Comparative Ophthalmology

Degradation of Motor Functions and of Electrical Reaction in Ocular Muscles of Birds. A. Rochon-Duvigneaud and C. Veil, Compt. rend. Soc. de biol. **121**: 825, 1936.

Though eye movements are fairly extensive in certain birds (hornbill, cormorant, pelican and tern) they are quite restricted in the Gallinae (chicken, pheasant, turkey, grouse, partridge and quail), and the eyes of owls are entirely immovable. In the latter groups the ocular muscles, though well developed and normally innervated, are attached posterior to the equator with a consequent loss of efficiency. The absence or deficiency of movement is compensated by extreme mobility of the head. In the pigeon and owl the ocular muscles are practically without excitability to electric stimulation. It is interesting that these muscles, though inert, nevertheless maintain their normal structure.

J. E. Lebensohn.

Cornea and Sclera

Familial Cornea Plana, Complicated by Cataracta Nigra and Glaucoma. H. Barkan and W. E. Borley, Am. J. Ophth. **19**: 307 (April) 1936.

Cornea plana in a mother and two daughters is reported. In one of the daughters the condition was complicated by glaucoma, and in the mother, by cataract. All showed absence of the normal curvature of the cornea, which in profile could be seen to have the same curvature as the sclera. Two of the patients had an opacity around the limbus, with blood vessels which extended about 1 mm. into the corneal tissue. A table of the corneal measurements and errors of refraction is given. The authors conclude that, clinically and practically, patients with cornea plana can be told that (1) the condition is familial; that is, if one child has it, other children will be apt to have it; (2) that, as it is probably a dominant inheritable characteristic, patients contemplating marriage should bear in mind that their children may have it; (3) that glaucoma may be a complication.

W. S. Reese.

Some Rare Forms of Keratitis: Superficial Neurogenic Keratitis; Familial Degeneration of the Cornea. G. E. de Sanctis, Ann. di ottal. e clin. ocul. **64**: 160 (March) 1936.

Two cases of the first condition are described. They occurred in women aged 25 and 53 years, respectively, who had attacks of pain

and inflammation for some months with reduction of vision, in the first case to 8/10 and in the second to 4/10. Slit-lamp examination after staining with fluorescein showed that the corneal epithelium was intact and that the stroma immediately under Bowman's membrane, especially in the central region, contained numerous minute white flecks. These were much more extensive in the second case. They were of various shapes, some punctate and others elongated into small striae or chains of points. There were some pigment granules on the epithelium but no inflammatory deposits. In both cases the cornea showed marked reduction of sensitivity. The condition was present in no other members of the family, and de Sanctis believes it to have been due to primary damage of the nerve endings. Other conditions described in the literature resembling this are discussed.

His third and fourth patients were a father and son showing an accumulation of grayish white dots through the entire depth of the cornea. The corneal epithelium was intact and was at no point elevated by the deposits. The corneal sensitivity was not affected. Many of the dots were highly refractile, resembling crystals. The condition resembled cases described by Fleischer as group I.

The article is furnished with a bibliography.

S. R. GIFFORD.

WHITE RINGS IN THE CORNEA. KUO PING KUAN, Arch. f. Ophth. **135:** 135 (Jan.) 1936.

The author adds eleven cases of his own to the twelve cases of white rings in the cornea reported in the literature (Uyamo, Yasuo: Concerning the White Ring in the Cornea, ARCH. OPHTH. **15:** 309 [Feb.] 1936). Clinically, the condition is that of white ring-shaped corneal opacities less than 1 mm. in diameter which are made up of fine granules and are approximately in the region of Bowman's membrane. Its cause is unknown.

P. C. KRONFELD.

Experimental Pathology

EXPERIMENTAL TUBERCULOUS PERIPHLEBITIS. Y. UYAMA, Arch. f. Ophth. **135:** 364 (May) 1936.

It is, on the whole, difficult to produce tuberculous periphlebitis of the retina. In most of the successful experiments a very big dose of bacilli was given, and the result was a severe uveal inflammation with involvement of the retinal veins. Uyama infected rabbits by intracardial injection of an emulsion of the bacilli in amounts of 0.01 mg. per kilogram of body weight. Some of these rabbits had been treated by repeated subcutaneous injections of tuberculin before their infection. Periphlebitis with slight choroiditis developed in four of twenty-six animals, while among the controls (receiving no treatment with tuberculin before infection) only one of fourteen showed periphlebitis. The pathologic result was the same in both groups, namely, small perivascular infiltrations. The anterior parts of the uvea and retina were normal.

P. C. KRONFELD.

General

THE RELATIONSHIP OF SPRING TO DISEASES OF THE EYES. A. JESS, Deutsche med. Wchnschr. **62**: 627 (April 17) 1936.

The populace has an idea that spring has a beneficial effect on diseases of the eye, that operations on the eye should be postponed until spring to obtain the best results. The results of intra-ocular operations such as those for cataract and glaucoma are not influenced by the season.

Naturally when speaking of spring and the eye the physician thinks of spring catarrh. A description of the signs and symptoms of this entity is given. The many theories as to the cause of the condition are discussed.

Another ocular disease noted frequently in spring and also autumn is rosacea of the conjunctiva and cornea. It is usually associated with a similar disease of the skin of the face. After briefly describing the condition Jess recommends an ammonium-ichthyol-zinc salve suggested by Peters for treatment. Diseases due to vitamin deficiency are often found during the early spring because of inability to get the necessary vitamins during the previous winter. Keratomalacia and hemeralopia are often seen in epidemics. The weather may be said to have little influence on diseases of the eyes.

L. L. MAYER.

General Pathology

THE RELATION OF VITAMIN A TO ANOPHTHALMOS IN PIGS. F. HALE, Am. J. Ophth. **18**: 1087 (Dec.) 1935.

Hale noted anophthalmos in all of a litter of eleven pigs of a gilt of the Duroc breed which had been fed a vitamin-free diet for one hundred and sixty days before breeding and thirty days after breeding. Since similar anomalies had never been observed in this herd it was assumed that the maternal deficiency of vitamin A had caused the ocular anomaly. A second experiment was unsatisfactory in that one gilt failed to show the symptoms of estrus, and the other failed to farrow, postmortem examination indicating that the litter had perished at an early stage and that this was followed by complete resorption of the fetuses. Another gilt which had gone through a one hundred and ninety day period without vitamin A had a litter of ten pigs all of which showed anophthalmos. A second gilt to which a 2 ounce (56.7 Gm.) dose of cod liver oil had been given on the one hundred and seventy-sixth day because of weakness produced a litter of fourteen pigs all of which were blind and showed combinations of ocular defects such as anophthalmos; some had one eye; some, one large and one small eye. In both litters there were other anomalies. Hale rules out the possibility of this being due to heredity by showing that on various matings of the blind pigs with each other and with normal pigs the offspring were all normal. These studies leave no reasonable doubt that a maternal deficiency of vitamin A will be expressed in a variety of defects in the offspring, including blindness, even failure of complete development of the eyes, cleft palate, harelip and arrested ascension of the embryo kidney.

W. S. REESE.

Hygiene, Sociology, Education and History

FUNCTIONAL LOSS AND WORKMAN'S COMPENSATION IN OCULAR INJURIES. A. CONTINO, Ann. di ottal. e clin. ocul. **64:** 73 (Feb.) 1936.

Contino raises the objection to previous systems of compensation that they fail to recognize properly the much greater importance of losses near the limits of industrial blindness as compared with losses of small degree. Most of these schemes, moreover, make no provision for functional losses other than that of central vision. He proposes a simple formula which he believes will allow an equitable indemnification under all circumstances. It is as follows:

$$D = 35/65\ P\ P^1$$

Here *D* represents indemnity, *P* loss of central vision and P^1 loss of other functions. Under the Italian workmen's compensation law, loss of one of two good eyes is estimated at 35 per cent of total disability, while loss of the only eye is estimated at 65 per cent. Thus for partial loss of vision in the first eye the formula would be $D = 35\ P\ P^1$ and for loss in the only remaining eye $D = 65\ P\ P^1$.

When, as usually happens, loss of central vision is accompanied by similar loss in other functions $P = P^1$ and the formula becomes $D = 35/65\ P^2$.

Cases are given to show the value of the formula. Loss of central vision in the first eye amounting to 5/10 when accompanied by similar loss in other functions would thus be $D = 35\ (5/10)^2 = 8.75$ per cent. If the loss were 9/10 it would be estimated as 28.35 per cent of total disability, the higher losses giving greater disability in proportion to their importance. The formula allows the use of test types recording fractions of any kind.

When a deficiency in vision is known to have existed prior to the accident it is calculated by the same formula and subtracted from the loss due to the injury.

Contino believes that the smallest amount of loss for which compensation is paid should not be 11 per cent, as proposed in a recent law, but 8 per cent.

S. R. GIFFORD.

Injuries

RARE PATHOLOGIC OBSERVATIONS AFTER OCULAR INJURIES. A. FUCHS, Klin. Monatsbl. f. Augenh. **95:** 721 (Dec.) 1935.

Fuchs reports the history of three unusual posttraumatic ocular conditions and gives the histologic observations.

1. The right eye of a man aged 48 was perforated by a shot. The shot recoiled from the posterior pole of the eyeball to the ora serrata, whence it was thrown back into the center of the eyeball, where the roentgenogram showed it to be located. A shred of epithelium was carried by the shot through the retina into the subretinal space near the disk. This epithelium spread on the choroid and surrounded a portion of the retinal vessels.

2. A woman aged 57 died from pneumonia four days after successful extraction of a cataract. The incision was closed only outside by fibroblasts and epithelium while inside it still gaped. Several giant cells had formed within four days in the center of the apparently well closed wound. They may have been caused to form by the presence of a small particle of cortical substance or of capsule of the lens or possibly by a cotton fiber as observed in a specimen of Salzmann's.

3. A retrobulbar tumor was removed with preservation of the eyeball in a woman aged 27. Chronic iritis with atrophy of the iris, formation of a pupillary membrane and development of a cataract followed. Recurrent inflammation necessitated enucleation for atrophy of the eyeball a year later. Histologic examination showed against expectation that the vitreous chamber was not filled with a hull and that the retina was not completely detached. Fuchs thinks that the iridocylitis was caused by degenerative processes at the posterior pole of the eyeball. The retina, which presented a thick mass of glial substance, was detached only at the periphery. Unusual, furthermore, was the presence of thin osseous platelets within the choroid, chiefly in its innermost layers. The main mass of the ossification rested like a shell on the remnants of the retina in the posterior pole of the eyeball; it originated by transformation of a hull on the retina without causing a detachment. Another unusual feature was an extensive proliferation of the pigmented epithelium at the posterior pole of the eyeball. This epithelium formed tubes and cell proliferations. The tubes had lumens, into some of which the blood had flowed. The heaped mass of epithelium gave this formation the appearance of an adenoma. The posterior ciliary vessels had not formed again after the removal of the tumor. Only two arteries were present. They entered the eyeball through the atrophied stump of the optic nerve.

K. L. STOLL.

Lacrimal Apparatus

TREATMENT OF EPIPHORA AFTER EXTIRPATION OF THE LACRIMAL SAC. B. G. TOWBIN, Arch. f. Ophth. **135:** 579 (July) 1936.

Towbin describes an operation which follows the method of Dupuy-Dutemps until the nasal mucosa is exposed through the hole in the bony lacrimal fossa. A nasal mucosa flap is formed with its base above the hole in the bone. Through this hole the flap is brought into the lacrimal fossa and in contact with the mucosa of the lower canaliculus. A thick suture is led through the canaliculus into the nose. The flap of nasal mucosa is wrapped around this thread and the wound closed. The heavy suture is left in situ for ten days. In one case, with an operation after this method Towbin effected a lasting communication between the lower canaliculus and the nose.

P. C. KRONFELD.

THE ACTIVE FUNCTION OF THE LACRIMAL TRACT DURING BATTING OF THE EYELIDS. B. ROSENGREN and I. KUGELBERG, Klin. Monatsbl. f. Augenh. **95:** 764 (Dec.) 1935.

Disagreement still exists regarding the mechanism of the conduction of tears through the tear passages, which possess no contractile elements. The only muscle connected with the tear passages is the

orbicular muscle, which forms sphincter-like loops around the tear points. Starting from this consideration, the authors carried on an extensive research by means of a small pump of their own device. A fine rubber tube was inserted into the tear point after it had been passed through a horizontal glass tube attached to a graduated plate. A small air bubble within the glass tube registered the motion of the fluid. This apparatus of registration was connected with a cinematographic mechanism of registration taking sixteen exposures per second. Photographs illustrate these devices, and eight tables record the observations made on seventy-eight persons with normal and pathologic lacrimal tracts. The authors made the following observations: The conduction of the tear fluid through the tear passages is accomplished chiefly by the active function of these passages. The active function observed during the experiments is identical with the physiologic function. It consists in a pumping action of the canaliculi: The lumen of each canaliculus is compressed during the batting of the eyelids and dilated between battings. Changes of the volume of the tear sac cannot physiologically eliminate the tear fluid from the conjunctival sac, but they are instrumental in facilitating the voiding of the lacrimal sac through the nasolacrimal duct.

K. L. STOLL.

Lens

DINITROPHENOL CATARACTS WITH SIGNS OF TETANY. E. B. SPAETH, Am. J. Ophth. **19**:320 (April) 1936.

Spaeth reports the case of a 37 year old nurse in whom cataracts developed following the use of di-nitrophenol as a weight reducer. Both lenses showed liquefaction which he believes would have eventuated in complete absorption had they not been operated on. Symptoms of tetany which cleared up on the administration of parathyroid suggested that the cataracts were due to the lack of this, though they did not look like the cataracts observed in association with tetany. Excellent operative results were obtained.

W. S. REESE.

INCIDENCE OF CATARACT AT CERTAIN AGE PERIODS IN SOUTH INDIAN DISTRICTS. R. E. WRIGHT, Brit. J. Ophth. **20**: 545 (Sept.) 1936.

The figures given by Wright in 1935 were obtained while he was conducting an inquiry into the possible etiologic influence of infra-red solar radiation on the production of cataract in two different areas where exposure to infra-red radiation might be expected to differ considerably. Two thousand outdoor workers between the ages of 40 and 60 were examined in a dry and arid district (Bellary); 1 in 5.1 had cataract. Two thousand outdoor workers were examined in a more humid and fertile area (West Coast); 1 in 3.4 had cataract. These figures obtained by trained observers include, of course, persons with immature cataract, even when this occurred on one side only. The sexes appeared to be affected to a like extent. The apparent age was frequently given rather than the actual age, since numbers of those examined had no idea of the latter. The majority probably looked older than they were. The figures will prove surprising to many.

W. ZENTMAYER.

EXTRACTION OF CATARACT IN ITS CAPSULE. L. COLLIN, Ann. d'ocul. **172:** 43 (Jan.) 1936.

Of all present day questions in ophthalmologic circles, none receives such an amount of discussion as that of extraction of cataract. Most operators tend now toward extraction of the lens in its capsule, although this operation began to be used only ten or fifteen years ago. In a report presented this year before the French Society of Ophthalmology, R. de Saint-Martin gave a history of this method. The operation appears simple but demands delicate execution. With a little experience and study all ophthalmologists may practice it with success. It is essential to bear in mind the two following matters: (1) anesthesia and akinesia and (2) mydriasis. Collin discusses the incision, the iridectomy, the grasping of the capsule, the disinsertion of the zonule and the extraction.

S. H. McKEE.

DINITROPHENOL. P. DALLY, Arch. Soc. d. sc. méd. et biol. de Montpellier **17:** 109, 1936.

Dally maintains that the complications resulting from the use of dinitrophenol are due to impurities. The cataracts produced are similar to experimental naphthalene cataract; they are probably due to dinitronaphthol, an impurity of the drug. The cases reported have all originated in the United States. In France no such complication has been noted.

J. E. LEBENSOHN.

HEREDITY IN SENILE CATARACT. M. BÜCKLERS, Klin. Monatsbl. f. Augenh. **96:** 119 (Jan.) 1936.

Two genealogies are presented showing members afflicted with senile cataracts in three consecutive generations. They were operated on and observed at the eye clinic of the University of Tübingen, Germany. Examinations made with the slit lamp during the last ten years showed the posterior shell shape type of cataract in one family and the nuclear and cortical cataract with radii in the other. It is doubtful if the consanguinity evidenced in these genealogies as far back as the seventeenth century is related to the frequent occurrence of the cataracts. The importance of what Vogt called the "senile determination" is pointed out in view of the still prevailing idea of the exogenous origin of senile cataract and of the possibility that it can be influenced by drugs.

K. L. STOLL.

Lids

PALPEBRAL DYSCHROMIA AND POLIOSIS OF THE CILIA. G. ANASTASI, Ann. di ottal. e clin. ocul. **64:** 59 (Jan.) 1936.

Four types of palpebral dyschromia and poliosis of the cilia are described and illustrated with typical cases. The type due to vitiligo affecting the lids and lashes is illustrated in the case of a man now

40 years old in whom the condition developed at the age of 7 and remained stationary. Both upper lids and an area in the temporal region were deprived of pigment; the cilia were unaffected. No systemic cause was found. The cilia have a later origin in intra-uterine life than the hairs of the brow and scalp and are almost never affected when the hair of the scalp undergoes senile depigmentation. The depigmentation of the lashes that is due to trophic changes is illustrated in the case of a patient with old trachoma in whom areas of the cilia became white. A similar condition resulting from local trauma is illustrated in the case of a man whose upper eyelid together with the cilia and brow became depigmented following injury in a fall. The type due to inflammation of the eye, associated with intense pain in the periocular region, was seen in a boy of 14 years who had suffered from bilateral iritis and articular rheumatism. The pupils were fixed by synechiae and the irides showed areas of depigmentation. In a zone surrounding the palpebral fissure almost completely on both sides the skin was depigmented, and many of the lashes were white. The possibility that atropine is a factor in the trophoneurotic disturbance which causes depigmentation must be considered on account of the report of Corrado.

S. R. GIFFORD.

Methods of Examination

THE USE OF THE FLICKER PHENOMENON IN THE INVESTIGATION OF THE FIELD OF VISION. L. A. RIDDELL, Brit. J. Ophth. 20: 385 (July) 1936.

The subject is considered under the following heads: I. Rationale of the Method. II. Difficulties in Using the Method. III. Arrangement of Apparatus. IV. The Value of Flicker in Investigating Visual Fields: A. Flicker in the Normal Subject. B. Observations in the Abnormal—(1) Cases of Intracranial Lesions. (2) The Effect of the Condition of the Retina upon Fusion Frequency.

Riddell states: "It seems undoubted that the flicker method is to have a place in ophthalmic work, whatever its deficiencies in the type of case studied in this paper. It is also probable that just as the method has been used to study such a fundamentally neurophysiological problem as inhibition, so it may be used in the study of changed neuronic responses in neuropathology, for instance in the Psychoneuroses."

He makes the following conclusions:

"*In cases of intracranial tumour*:—(1) Campimeter fields and flicker fields in general agree.

"(2) Neither is the more sensitive; flicker will show no defect that cannot also be found with careful campimetry; and moreover flicker results are much more difficult to interpret, owing to their greater dependence upon external conditions such as lighting, and upon age, etc.

"(3) Flicker cannot show contours as in campimetry.

"(4) Flicker may, however, be used to estimate the density of scotomata with fair accuracy. It may also serve to indicate if the fixation point is especially involved in cases that show a lowered visual acuity.

"(5) The flicker method is at least as difficult as campimetry, and the technique is just as difficult to learn. One set of conditions should be decided upon and adhered to, as otherwise calibratory control cases, which are essential, become invalid.

"(6) The apparatus recommended is a compact one (C III of the Appendix). If it is hoped to *detect* field defects, the flicker should be of the same intensity as the screen: but if *known* scotoma are to be measured, a brighter light may be easier to use.

"In other conditions:—The method will probably find greater application in purely ocular conditions, as suggested by the work of Granit, and as successfully used already by Teräskeli and by Enroth and Werner.

"It may also prove of use in some purely neurological conditions."

W. ZENTMAYER.

Neurology

THE PIGMENT RING OF KAYSER-FLEISCHER IN PSEUDOSCLEROSIS. J. FROMENT, P. BONNET and L. PAUFIQUE, Bull. Soc. d'opht. de Paris, November 1935, p. 713.

The diagnosis of the patient's general condition was made from the findings in the cornea. On direct examination the pigment ring was poorly visible, but on oblique illumination it was observed on the posterior surface of the cornea near the limbus. There was no interference with the conjunctival circulation. On the posterior surface of the cornea a ruby red circle concentric with the limbus faded into a verdant green. There were in reality two rings, separated by an area of clear cornea. Because this double ring is unusual and similar to that in Kestenbaum's case, the authors felt it worthy of report.

L. L. MAYER.

METHODS IN THE DIAGNOSIS OF TUMORS OF THE BRAIN. P. HILPERT, Klin. Monatsbl. f. Augenh. **95:** 577 (Nov.) 1935.

Hilpert describes the modern methods employed in the diagnosis of tumors of the brain at the neurologic clinic of Greifswald, Germany. Discussing these methods, he quotes the literature in point and adds eighteen roentgenograms. This paper is exhaustive, containing much valuable information to which justice cannot be done in an abstract.

Aside from the methods used in the clinical diagnosis of tumors of the divers portions of the brain and medulla, the author delineates the following accessory methods:

1. Tapping of the spinal fluid, examination of the fluid and registration of the intracranial pressure.

2. Roentgenography, important for changes in the sella turcica, for calcifications that may occur in connection with tumors, for changes in the os petrosum, especially at the internal acoustic meatus in the presence of tumors of the acoustic nerve, for conditions producing pressure at the convexity of the skull, especially at the parietal bone; for those producing absorption of the bones of the skull, and for bony hyperplasias connected with meningiomas. Roentgenograms should be taken prior to air inflation of the spaces containing cerebrospinal fluid.

3. Tapping of the brain after Pfeifer's method, which has been more or less replaced by encephalography.

4. Encephalography and ventriculography. The technic and the indications for both methods are described in detail. Lumbar and suboccipital air inflations, their use and errors arising from their use are given consideration; examples of encephalograms and ventriculograms are added.

5. Arteriography after the method of Moniz is referred to. Hilpert states that his experience in this matter is insufficient to permit him to express a personal criticism.

These accessory diagnostic methods do not minimize the importance of an exact anamnesis. The first symptoms furnish the most reliable indication as to the location of a process which is limiting space, such as a growing tumor.

K. L. STOLL.

Parasites

OCULAR FILARIASIS: CONJUNCTIVAL LESIONS IN A PATIENT WITH ONCHOCERCIASIS CONTRACTED IN THE IVORY DISTRICT. C. JOYEUX, J. SEDAN and J. ESMENARD, Ann. d'ocul. **173**: 100 (Feb.) 1936.

The purpose of this paper is to describe the clinical signs of the ocular lesions, especially those of the conjunctival lesions, which enabled the authors to discover the filariasic origin of the patient's disease.

Appelman of Louvain has published a splendid article on the visual disturbances associated with African onchocerciasis. As this condition had not previously been reported in French Africa, although known there, the authors thought the observations on a patient who contracted the infection in the Ivory District would be of interest. The case is presented in considerable detail. That the ocular lesions were of primary importance is evidenced by the fact that the patient first sought an ophthalmologist for relief. These lesions were represented by subconjunctival granules with integrity of the cornea and transparent parts. The microfilariae were not observed on biomicroscopic examination, nor were they found in the fragments of conjunctiva excised with the granulomas. A prominent complaint was that of a condition very similar to the allergic reaction called "filariasis itch."

There are two illustrations and a bibliography.

S. H. MCKEE.

Pharmacology

A PHYTOPHARMACOLOGICAL APPROACH TO SOME OPHTHALMOLOGICAL PROBLEMS. D. I. MACHT, Am. J. Ophth. **19**: 324 (April) 1936.

Macht has studied the effects of drugs and poisons on living animal and vegetable protoplasm. These studies have led to the development of a department of science called "phytopharmacology." It was found that living biologic test objects from the plant kingdom are often much more sensitive to the effects of chemical agents than living zoologic test objects. He demonstrated the presence of various toxic substances

in human blood by using sensitive seedlings of Lupinus albus, kept alive in physiologic solution of sodium chloride. Macht claims that with the blood serum of patients suffering from pemphigus and trachoma a definite phytotoxic reaction is obtained which is of diagnostic value. Such a reaction indicates a systemic toxemia in these patients.

W. S. Reese.

The Mechanism of the Vasodilator Action of Atropine. Raymond-Hamet, Compt. rend. Soc. de biol. **122:** 42, 1936.

In toxic doses atropine not only induces intense vasodilatation in the skin but may even excite erection. Physiologic analysis indicates that atropine affects either the smooth muscle directly or the terminal innervation.

J. E. Lebensohn.

The Pupil

A Little Known Pupillary Phenomenon Observed During Drop Narcosis. A. Heinrich, Klin. Wchnschr. **15:** 1010 (July 11) 1936.

If one notices the pupillary reaction during narcosis, especially during ether anesthesia, one observes that the pupil fails to react to light when the anesthesia deepens. The specialist in narcosis Adolph Hets observed that if both eyes are opened during this stage and a light stimulus is applied the pupils show a reaction. When the narcosis becomes very deep this phenomenon is also lost. Heinrich explains the phenomenon on the basis that, the stimulus being greater when both eyes are lighted, the Edinger-Westphal nucleus is not so under the influence of the anesthesia that the augmented stimulus cannot cause a reaction. Hets and Strassman in 1894 and Czempin in 1897 called attention to this phenomenon, and in 1901 Flockemann showed the practical application. However, it is still little taken into account, and Heinrich wishes to call it to attention again. The article is supplied with a bibliography.

L. L. Mayer.

Physiology

The Circulation in the Retina Under Physiologic Conditions. J. Sobański, Arch. f. Ophth. **135:** 372 (May) 1936.

Sobański is a specialist in ophthalmodynamometry and has contributed materially to the improvement of its technic and toward proper interpretation of the values obtained with it. In the paper under review he sums up his findings in persons who were without evidence of any cardiovascular or ocular disease. He has used Bailliart's ophthalmodynamometer and the curves of Bailliart and Magitot to determine the pressure in the retinal arteries and veins. He states that in subjects over 15 years of age the average for the systolic arterial pressure is between 80 and 90 and the average for the diastolic arterial pressure between 48 and 56. These values are considerably higher than those found by

Bailliart. Sobański believes that there are pulsatory fluctuations of the pressure in the capillaries and in the veins also. He assumes that the capillary systolic and diastolic pressures in the retina are, on the average, 55 and 33.

He has found spontaneous venous pulsation, synchronous with the pulse of the carotid artery, in 40 per cent of his subjects and interprets it as a sign that the diastolic venous pressure is lower than the intra-ocular pressure. In those without spontaneous venous pulsation the diastolic venous pressure is higher than the intra-ocular pressure. In these the intra-ocular pressure has to be raised by compression to the level of the diastolic venous pressure (just as in determining arterial pressures by sphygmomanometry) to produce visible venous pulsation.

The most important fact about retinal circulation is that the central vein during its course through the intervaginal space is exposed to the intracranial pressure and that therefore the retinal venous pressure must be higher than the intracranial pressure. The retinal venous pressure on the other hand, is not influenced by arterial hypertension or by ocular hypertension. In glaucomatous eyes he has found the same venous pressure before and during a rise in intra-ocular pressure.

The retinal arterial pressure is closely connected with the general arterial pressure, the retinal venous pressure and the intra-ocular pressure. Disturbed venous outflow always makes the diastolic arterial pressure rise too.

All vascular pressures in the retina show a tendency to rise with increasing age.

P. C. KRONFELD.

Refraction and Accommodation

ANISEIKONIA. E. J. LUDVIGH, Am. J. Ophth. **19:** 292 (April) 1936.

This article does not lend itself to abstracting. Ludvigh points out that differences in the size of the retinal images occur normally in reading. He suggests that in the reported cases the symptoms may have been of psychoneurotic origin, and concludes that the contention of the existence of aniseikonia other than that due to obvious physical optical causes is based on inconclusive evidence and furthermore derives little support from theory, since it has not been demonstrated that the patients' complaints are attributable to the condition with which the theory deals.

W. S. REESE.

VARIATION OF THE REFRACTION AND CAMPTOPSIA (CURVED VISION) IN THE DIABETIC SUBJECT. R. ONFRAY and VIGOUROUX, Bull. Soc. d'opht. de Paris, December 1935, p. 772.

Since insulin began to be used in the treatment of diabetes, hyperopic changes in refraction have been reported, whereas previously myopia was the usual finding. These changes have been demonstrated to be due to variations in the osmosis of the lens. The case which the authors report is similar to those of Spalding, Post, Berke and Dickmann and others except that a newly found phenomenon was revealed as a result of intelligent observation by the patient. After insulin therapy hyperopia developed with a subjective curved distortion of the images seen by the

patient, the right eye noting a bowed out curve while objects seen by the left eye were bowed or curved inward. Two months later, when antidiabetic treatment was discontinued, the phenomenon disappeared. A complete history of the case is outlined, and drawings by the patient show the distortion. The authors feel that osmotic changes of the deeper structures of the lens, on one hand, and such changes in the peripheral parts of the lens, on the other hand, may account for the difference in curve.

L. L. MAYER.

Retina and Optic Nerve

HISTOLOGY OF A RECENT DETACHMENT OF THE RETINA. BAILLIART, MAWAS and TILLÉ, Bull. Soc. d'opht. de Paris, December 1935, p. 761.

Examined two days after a sudden loss of vision, the left eye presented a detachment of the retina measuring 15 diopters at its summit between the meridians of 12 and 5 o'clock, involving the interpapillomacular region. Tears were found situated at 1 o'clock at the ora serrata measuring one-fourth disk diameter and one between 2 and 3 o'clock, 5 disk diameters from the ora serrata. Diaphanoscopy aroused suspicion of a tumor, and enucleation was advised. At the time of enucleation the detachment was known to have existed for twenty-two days. The entire retina was found to be detached, plaited and wrinkled, very thin and lacelike.

A summary of the microscopic examination follows: There was sclerosis of the choroidal arteries, with hyaline degeneration and thrombosis of several branches. An insufficiency of circulation was indicated by the edema with detachment of the choroid and ciliary body. There were important lesions in the pigmented epithelial layer, consisting of hypertrophy and hypersecretion of pigment and the formation of an exudate beneath the retina. The retinal lesions consisted of atrophy with degeneration of cystic formations, tears and pyknosis of the cells with visible cytolysis in many layers.

The authors believe that the vascular lesions of the choroid caused changes in the retinal pigmented layer with cystic formation and retinomalacia and thus account for the detachment. Excellent photomicrographs are included.

L. L. MAYER.

RIGIDITY OF THE CENTRAL ARTERY OF THE RETINA. J. A. PEYRET, Arch. de oftal. de Buenos Aires **10**: 443 (June) 1935.

Peyret refers to experiments carried out by Fritz under Coppez' direction in Belgium on the measurement of the rigidity of the central artery of the retina with Bailliart's ophthalmodynamometer. He explains Fritz' index (with a normal mean resistance of 10 Gm.), distinguishing four types of pathologic retinal circulation: (1) normal or reduced softness of the wall of the vessel with diastolic hypotension and normal or narrowed caliber of the retinal arteries, showing poor renal function; (2) lowered arterial softness (resistance under 10 Gm.) with diastolic hypertension, the caliber of the arteries being normal or slightly

dilated; (3) excessive rigidity (resistance over 20 Gm.) with normal or reduced diastolic tension and marked arterial narrowing and rigidity; (4) increased systolic and diastolic tension with normal caliber of the vessels.

The importance of determining the index is great if taken in relation to the arterial tension in the brachial artery in cases stressed by Coppez as follows: (1) cases of cerebrospinal hypertension, ventricular dropsy and cerebral commotion, in which the brachial tension is normal with retinal hypertension and a normal Fritz index; (2) cases of cardiorenal disease and lesions of the retinal vessels with general arterial lesions and variations in tension according to the particular disturbance; (3) cases of cerebral or retinal angiospasm, the data on which are confusing, although in that limited to the retinal system there is some local excess of rigidity of the vessels with corresponding visual disturbances and in that limited to the cerebral vessels the retinal vessels show normal rigidity.

Peyret describes the findings in sixteen cases in detail.

C. E. FINLAY.

HYALINE EXCRESCENCES OF THE OPTIC DISK. F. J. SORIANO and H. R. PICOLI, Arch. de oftal. de Buenos Aires **10:** 685 (Sept.-Oct.) 1935.

This is the report of two cases of hyaline excrescences of the disk, in one markedly exuberant and in the other limited to four spherical bodies. In the first, although central vision was normal, there was a marked fan-shaped defect in the upper part of the visual field, starting from the blindspot.

C. E. FINLAY.

OPERATION FOR RETINAL DETACHMENT. F. BELGERI and M. DUSSELDORP, Arch. de oftal. de Buenos Aires **10:** 751 (Sept.-Oct.) 1935.

Thirty patients with retinal detachment were operated on according to Arruga's procedure: denudation of the sclera over the area of the tears, scorching of the scleral surface at this spot in a 10 to 15 mm. circle with a ball electrode and a current of 100 milliamperes, six punctures within this circle with a Lacarrère or Šafář point and central galvanopuncture, followed by suture of the conjunctiva and overlying tissues. They insist on the necessity after the operation of absolute rest for twenty days with the head in such a position that the tear is at the most dependent portion of the eye, followed by the use of a stenopaic slit during some time, with the object of favoring immobility of the eye. Their results were: fifteen successes and fifteen failures.

C. E. FINLAY.

DOSAGE IN THE TREATMENT OF RETINAL DETACHMENT BY ELECTROCOAGULATION (DIATHERMY). A. MOREU, Arch. de oftal. hispanoam. **35:** 460 (Sept.) 1935.

Moreu calls attention to the empyric dosage of cauterization in the procedures employed in the closure of retinal tears (Gonin, Lindner-

Guist, Weve, Šafář, Larson, Meller) and gives illustrated reports of two cases in which a primitive closure of a tear was followed by one produced by cicatricial contraction due to excessive cauterization in the first operation. He then stresses the importance of regulating the temperature in the diathermic procedures, which he prefers, and after showing the defects of Coppez' pyrometric electrode describes a regulating electrode which he has devised, based on Regaud's electric regulator.

The procedure which he recommends consists in a precise localization of the tear or tears, the usual preparatory operative steps, with baring of the overlying sclerotic, the application of a platelike electrode, 2 mm. in diameter, at a temperature of 80 C., producing a series of nonpenetrating cauterizations at the site of the tear and making, finally, two or three punctures with a Šafář-like electrode at a temperature of 60 C. The effect can be further controlled with the ophthalmoscope. The succeeding steps are similar to those of other procedures.

C. E. FINLAY.

DETACHMENT OF THE RETINA AND ACCIDENT. C. ZENKER, Klin. Monatsbl. f. Augenh. **95**: 393 (Sept.) 1935.

Zenker discussed the question of detachment of the retina and accident from the standpoint of industrial compensation at a meeting of the Bavarian Ophthalmologic Society at Munich in June 1935. Detachment of the retina may be considered as a consequence of an accident if a direct connection has been established objectively; if it occurred, for instance, after a penetrating wound or in association with hematoma of the eyelids or with hemorrhage in the anterior chamber after contusion. It is immaterial whether the detachment occurred directly after the accident, later or after extraction of a traumatic cataract. Dehiscences of the ora serrata following contusion occur frequently in the upper portion of the fundus, whereas the spontaneous ones are frequently seen in the temporal or the lower portion, according to Gonin.

The etiologic decision is more difficult if an accident is supposed to have caused the detachment indirectly in the absence of any objective observation of consequences of the injury. Predisposition toward detachment by reason of myopia or age may play a rôle in this group; in this light must be considered also cystoid degeneration and atrophy of the retina, synchysis of the vitreous, rarefaction of the sclera (Guist), anomalous insertion of the superior oblique muscle (Peters) and mechanical moments related to the eyeball as a moving organ. The accident may be recognized as the cause of the detachment only if the latter can be traced as a direct consequence of the accident; the original statement made by the patient during the first visit is of importance.

Indirect lesions such as occur through concussions of the head or body or falls may cause detachment; predisposition may have existed. Zenker recognizes "excessive increase of commonly performed occupational strain" as another cause of detachment, which is contrary to the views of von Hippel and zur Nedden. Heavy labor is known to have produced tears in the retina or choroid of emmetropic eyes. To disallow such claims would not be justifiable.

In the discussion of this paper Schieck agreed with Zenker and regretted that zur Nedden's view was accepted by the Board of Compensation. Passow, after giving a definition of the legal term "accident," admitted that excess strain may be accepted as the cause of detachment under certain circumstances. Denying that bodily strain may be a primary cause of detachment, he conceded that detachment may follow excess strain or a fall, adding, however, that he had observed that the eyes of this group were predisposed by myopia of high degrees.

K. L. STOLL.

RETROBULBAR NEURITIS, DETACHMENT OF THE RETINA AND PARESIS OF THE EXTERNAL RECTUS MUSCLE AS INITIAL SYMPTOMS OF POSTERIOR SCLERITIS: REPORT OF A CASE. O. KARASEK, Klin. Monatsbl. f. Augenh. **95:** 645 (Nov.) 1935.

A woman aged 32 was referred to the hospital because of headache, nausea, conjunctivitis, diplopia (predominating prior to the menses) and optic neuritis. In addition to these symptoms, others were found in the right eye at the hospital: paresis of the external rectus muscle of the right eye as the source of the diplopia, an absolute central scotoma for all colors, a broadened blindspot and papilledema. The roentgenogram of the skull showed slightly increased intra-ocular tension and an osteoma in the left frontal sinus. The papilledema increased, and a week later the retina became slightly detached in the macular region. After another week the sclera near the temporal canthus became slightly prominent and tender. The symptoms, including the macular detachment, receded slowly and disappeared after about three weeks.

Previous reports of similar conditions are scarce and the symptoms described varying, rendering the diagnosis of posterior scleritis difficult. Subjective uneasiness of the eyes and pain on their motion may be associated with no manifestions of involvement of the anterior segment of the eyeball and with no change in the fundus except flat detachment of the choroid and retina. The temporal periphery of the retina and the macular region are involved most frequently. The inner ocular membranes are usually free from inflammation. The presence of optic or retrobulbar neuritis renders the diagnosis more difficult, and in some cases this will be possible only as the disease progresses. The diplopia which occurs rarely in this connection may be the result of a toxic or infectious lesion of the nerve of some extrinsic muscle. The exacerbation preceding menstruation may be explained from this point of view.

K. L. STOLL.

Trachoma

TRACHOMATOUS CONJUNCTIVITIS. A. F. MACCALLAN, Brit. M. J. **1:** 635 (March 28) 1936.

This is an abstract of the Hunterian lecture, "The Surgery and Pathology of Trachomatous Conjunctivitis," which MacCallan recently delivered at the Royal College of Surgeons of England.

He states that knowledge of the etiology of trachoma is incomplete, that it may be impossible to diagnose the disease clinically in the absence of bulky and expensive apparatus, that no advantages are

obtained by chemical or microscopic tests and finally that treatment of trachoma has not improved during the past thirty years. He then carefully considers the pathology and summarizes his views as follows:

"First, the primary epithelial lesion caused by the virus results in an inflammatory reaction in the subepithelial tissue, exhibited by a lymphocytic infiltration. . . .

"Secondly, this lymphocytic invasion of the subepithelial tissue spreads from its area of inception in the tarsal conjunctiva to the upper fornix, and thence in the bulbar conjunctiva to the corneal limbus, where changes occur peculiar to trachoma.

"Thirdly, the bleb-like excrescences, which exude a gelatinous material when ruptured, have no relation to the so-called trachoma follicles of an earlier stage of the disease.

"Fourthly, the lymphocytic invasion of the subepithelial tissue penetrates the tarsus, and finally is resolved into connective tissue. Hence result the deformities of the lids which occur in no other form of conjunctivitis."

MacCallan agrees with Mikhail that the changes at the upper corneal limbus occur by direct extension of the infection of the subepithelial tissues and that the limbus does not become infected from the trachomatous conjunctiva of the upper lid by contact.

Clinically he distinguishes the following four stages:

"First Stage.—After an incubation period of from four to ten days" a subepithelial infiltration appears which is concentrated into tiny grayish islets, the so-called follicles; "these form slight roughnesses, and first appear at the extremities of the tarsal conjunctiva at its upper border. The virulence of the trachoma virus is such that the subepithelial infiltration rapidly extends to the conjunctival fornix, and from thence to the upper corneal limbus. Pannus now appears. In the first stage of trachoma pannus is present as soon as the condition can be diagnosed; in fact the diagnosis rests on detecting pannus with the corneal microscope.

"Second Stage.—This has been divided into subgroups for application to different clinical appearances which are common in the East. For present purposes one subgroup only need be mentioned. Here there are numerous bleb-like excrescences, which rupture on pressure, with emission of gelatinous material. These are dilated ducts of sebaceous or meibomian glands. They have no relation to the tiny follicles of the first stage of the disease.

"Third Stage.—This shows the resolution of the inflammatory infiltration commencing and its replacement by scar tissue. Typically there are islands of inflamed conjunctiva surrounded by a cicatricial network. There is always some thickening of the tarsus by inflammatory products, often leading to entropion.

"Fourth Stage.—At this point all inflammatory changes have come to an end. The tarsus is covered by scar-tissue epithelium, and there is more or less cicatricial change in the thin subepithelial tissue and in the tarsus."

As for treatment, it is questionable whether the lymphocytic infiltration in the first stage should be destroyed since it is a defensive mechanism of the body and since no chemical or biologic agent has

been shown to have any neutralizing effect on the activity of the disease. The bleblike excrescences in the second stage should be rid of their secretion, and in the third stage the application of an irritant such as copper sulfate helps. Individual treatment by an experienced ophthalmologist gives the best results with a minimum amount of cicatrization.

The principal sequelae are trichiasis and trichiasis plus entropion, and they are best corrected, when an operation is necessary, by Streatfield's or by Van Millingen's operation.

A. KNAPP.

SURGERY AND PATHOLOGY OF TRACHOMATOUS CONJUNCTIVITIS. A. MACCALLAN, Rev. internat. du trachome **13**: 1 (Jan.) 1936.

Trachoma is widely spread among the Hindus, Mongolians, Malayans, Red Indians and Semitic peoples. The onset is insidious, and acute symptoms do not develop until roughness of the superior palpebral conjunctiva supervenes. The pathologic process consists of primary subepithelial lymphocytic exudation, in which the tarsus is involved, with secondary connective tissue metaplasia. The pannus is an extension of the cellular exudate between the corneal epithelium and Bowman's membrane; it is not a contact infection from the trachomatous conjunctiva. Peritomy for it is useless.

The disease has four stages: 1. After an incubation period of from four to ten days the subepithelial infiltration appears and is usually concentrated in tiny islets (follicles). Extension of this process into the cornea produces the pathognomonic pannus. 2. The meibomian ducts, strangulated by the scar tissue, form bleblike excrescences containing gelatinous material (the so-called follicles of the second stage). 3. A cicatricial network surrounds islands of inflamed conjunctiva. The thickened, deformed tarsus inclines to entropion. 4. All inflammatory changes are resolved. The tarsus, involved in scar tissue, is covered by scar tissue epithelium.

No treatment will abort the disease in stage 1. In stage 2 the blocked ducts should be emptied by mechanical means. In stage 3 application of irritants favors the deposition of fibrous tissue. Contraindicated in surgical manipulations are epilation, removal of skin and the use of lid clamps.

J. E. LEBENSOHN.

Tumors

A NEW WAY TO DETERMINE THE PROGNOSIS OF UVEAL SARCOMA. E. VON HIPPEL, Arch. f. Ophth. **135**: 79 (Jan.) 1936.

Freund and Neuberg found that the serum of persons not affected by malignant tumors has a histiolytic property for cells of such tumors, while the serum of patients with sarcomas or carcinomas lacks this property. Recently Klein (*Wissensch. Woche zu Frankfurt-a.M.* **2**: 39, 1935) modified the technic of Freund and Neuberg and reported his results. Von Hippel now recommends this method as an aid in determining the prognosis of uveal sarcoma. The evidence of the presence

of histiolytic properties is called "a negative Klein reaction" (meaning that the patient has defensive power against neoplastic growth and therefore no tumor); the evidence of the absence of histolytic properties is called "a positive Klein reaction." The patient with uveal sarcoma is expected to give a positive Klein reaction before the enucleation and to give a negative Klein reaction afterward if no metastases are present. The method is very delicate, and the possible sources of error are multiple. Von Hippel had the serums of twenty of his patients with uveal sarcoma examined by Klein himself. Of those patients, twelve who were living and well from six to twenty-nine years after enucleation of the sarcomatous eye were reported "Klein-negative" as was expected; however, the serums of three patients living and well from eighteen to twenty-five years after enucleation of the sarcomatous eye turned out to be "Klein-positive" against von Hippel's expectations. He recommends Klein's method for general use.

P. C. KRONFELD.

Uvea

CHANGES IN THE WALL OF THE LARGE CIRCULAR ARTERY OF THE IRIS IN DIFFERENT FORMS OF UVEITIS. J. MELLER, Ztschr. f. Augenh. **87**: 239 (Nov.) 1935.

The studies are limited to primary changes in the wall of the vessel, which in the normal eye maintains an open lumen. Only spontaneous chronic iridocyclitis, posttraumatic iritis and sympathetic iridocyclitis are included. The normal histology is given. Pathologic specimens from patients in whom the conditions observed were typical are studied in detail, with pathologic illustrations and case histories. Meller feels that these diseases are endogenous and that the walls of the vessels through which the various agents must pass should show similar changes. All three conditions showed similar primary changes. In most cases the endothelium showed proliferation with closure of the lumen and formation of a heap of epithelioid cells. The muscularis was thickened by proliferation of its cells to form an "onion peel" structure. There were fresh infiltrates in the media, and the external elastic layer was either absent or greatly thickened. The adventitia was infiltrated by plasma cells and lymphocytes, but only the plasma cells were considered as evidence of primary disease. Thrombosis was not found. One case of spontaneous iridocyclitis in which posttraumatic iritis developed after extraction of a cataract proved to be one of typical sympathetic disease; the characteristic findings were seen in the circular artery of the iris, indicating an endogenous sympathetic disease. The probability that tuberculosis was the causative agent is discussed in detail. There was much evidence to show that the toxic agent had been acting for a considerable time before the clinical signs of disease made their appearance. It was felt that the nature of the toxic agent could not be determined from histologic studies, but the endogenous character and the route to the iris were definitely established.

H. GIFFORD JR.

Vision

SCIENTIFIC AND PRACTICAL CONSIDERATIONS INVOLVED IN THE NEAR-VISION TEST WITH PRESENTATION OF A PRACTICAL AND INFORMATIVE NEAR-VISION CHART. J. E. LEBENSOHN, Am. J. Ophth. **19:** 110 (Feb.) 1936.

Lebensohn considers the essential weakness of present reading charts due to their oversimplification and to the fact that a single chart is being used to meet diversified scientific and practical demands. Two distinctly different charts are requisite, one containing simple optotypes based on the one minute visual angle and the other a standardized reading test in the various sizes of customary print. He presents two charts: The first contains an explanatory paragraph, optotypes in two series, letters and numbers, and a staff of music. The second has three columns presenting, respectively, words in point type, numbers of corresponding size and an illiteracy test. He describes how these charts are used to determine (1) the visual acuity for near objects, (2) the far point of myopia and (3) the range of accommodation and to obtain an estimation of visual acuity as a substitute for the distance test. When these charts are used in conjunction with the correction for distance they uncover malingering, visual efficiency, etc. W. S. REESE.

Vitreous

TUBERCULOUS NODULE FREE IN THE VITREOUS. J. A. SEÑÁ, Arch. de oftal. de Buenos Aires **10:** 419 (June) 1935.

A bright spherical body, the size of a rice seed, floated in the vitreous, and there was some secondary iridocyclitic and retinochoroidal reaction. The floating body was primarily diagnosed as a cysticercus. On failure in an attempt to remove the body, the eye was enucleated, and histologic examination revealed an encapsulated structure in which three zones could be distinguished: an outer rarefied, slightly chromatophilic zone in which some giant cells were apparent, a middle condensed cellular zone, and an internal zone in which besides cellular elements an interstitial substance with affinity for hematoxylin was observable. No bacterial elements were found. The neighboring vitreous was fibrillar and infiltrated with lymphocytes, as was also the adjacent retina, where cellular infiltration was especially marked in the vicinity of a retinal vein (periphlebitis). There was also slight lymphocytic infiltration of the base of the iris and of the ciliary body. The paper is illustrated with a number of cuts. C. E. FINLAY.

SLIT-LAMP STUDIES OF THE ANTERIOR BORDER LAYER OF THE VITREOUS AFTER INTRACAPSULAR EXTRACTION. L. SALLMANN, Arch. f. Ophth. **135:** 602 (July) 1936.

Senile cataracts were extracted after the Stanculeanu-Török method with the capsule forceps of Arruga, and the shape of the anterior border layer of the vitreous was studied. Usually the vitreous not only occupied the space previously taken up by the lens but protruded somewhat into

the anterior chamber. In some aphakic patients with choroidal detachment Sallmann found during certain periods a concave anterior border layer instead of the usual convex one. This phenomenon was visible when the choroidal detachment showed signs of regression, and apparently only if the regression took place fairly rapidly.

In one case secondary glaucoma following intracapsular extraction of a senile cataract required cyclodialysis twice. As long as cyclodialysis controlled the glaucoma, gentle massage of the eye produced at first increased protrusion of the anterior border layer of the vitreous, followed later by recession far beyond the original position. When the tension rose again after cyclodialysis, the anterior border layer lay behind the pupillary plane and hardly moved on application of massage. Sallmann interprets these observations as signs of outflow of intraocular fluid into the suprachoroidal space, in a channel which was opened by the cyclodialysis.

P. C. KRONFELD.

Sympathetic Ophthalmia

SYMPATHETIC OPHTHALMIA. A. C. WOODS, Am. J. Ophth. **19**: 9 (Jan.) 1936.

This interesting article does not lend itself to abstracting. Sympathetic ophthalmia is taken up under the following headings: history, definition, incidence, predisposing causes in the exciting eye, clinical picture in the exciting and in the sympathizing eye, interval between injury and onset, and duration of the disease.

W. S. REESE.

MENINGEAL REACTION IN SYMPATHETIC OPHTHALMIA AND IN TRAUMATIC INJURIES OF THE EYE. C. ESPÍLDORA-LUQUE, Arch. de oftal. hispano-am. **36**: 90 (Feb.) 1936.

Espíldora-Luque calls attention to the relative frequency of intense headache accompanied by vomiting in connection with sympathetic ophthalmia. At times there is papillary congestion. He considers these disturbances not as reflex phenomena but as suggestive of meningeal involvement. In support of this view he reports two cases of sympathetic ophthalmia in which lumbar puncture revealed a marked inflammatory condition, two in which such an inflammatory condition was present but less pronounced, and one in which it was very slight.

He does not consider the symptoms described as limited to sympathetic ophthalmia; they are also occasionally present with other varieties of uveitis (Gilbert's meningo-encephalismus). He gives the results of the examination of the spinal fluid in a case of intense uveitis.

In contrast he reports four cases of posttraumatic lesions without sympathetic ophthalmia, in which the results of examination of the spinal fluid were negative. He suggests examination of the spinal fluid as affording a possible prognostic sign of the production of sympathetic ophthalmia in traumatisms of the eye.

C. E. FINLAY.

Therapeutics

TUBERCULIN THERAPY IN TUBERCULOUS DISEASES OF THE EYE. A. Y. SAMOILOV and A. B. KATZNELSON, Sovet. vestnik oftal. **8:** 655, 1936.

This is a thorough and extensive study by an authority (Samoilov). The article is divided into (*a*) a theoretical part, (*b*) a clinical part and (*c*) basic conclusions. It is abstracted with difficulty. The authors divide tuberculosis of the eye into:

1. Metastatic, hematogenous tuberculosis, which is characterized by choroidal lesions and less frequently by disturbances of the iris and ciliary body. In most of the cases the process in the lungs is inactive. Two hundred patients of this group were treated with tuberculin. Samoilov observed in the choroiditis a reaction to the therapy in the form of movement of the pigment dust in the choroidal lesion, which indicated an activation of the process. He was thus guided as to the dosage of tuberculin.

2. Phlyctenular or, as Samoilov calls it, allergic keratoconjunctivitis, which is usually accompanied by an active bronchopulmonary process. One hundred and fifty patients were treated. The method of treatment in metastatic choroiditis was a gradual increase of tuberculin to the highest "anergyzing" or immunizing doses in order to cause a mild focal reaction. In allergic keratoconjunctivitis a prolonged desensitization of the organism and of the eye was obtained by initial high doses, as small doses might aggravate the exudative process.

Samoilov's basic conclusions are: The therapeutic rôle of tuberculin is different in metastatic and allergic tuberculosis of the eye. In the first condition it causes a specific focal reaction, while in the other it aids the desensitization of the organism. Tuberculin therapy is indicated in severe tuberculosis of the eye when routine measures are of no avail. In metastatic tuberculosis of the eye, because of the diffuse action subcutaneous injections are the method of choice, while in allergic tuberculosis of the eye intracutaneous or cutaneous injections are preferable, as they act directly on the skin, which is related to the eye. The dosage of tuberculin in metastatic tuberculosis depends on the activity and localization of the ocular lesion. In fresh lesions of the choroid weak solutions (1 : 10,000,000) are preferred, while in lesions of the anterior portion of the eye dilutions as strong as 1 : 100,000 can be applied. The sensitivity of choroidal lesions is so high that if there is no reaction to a dilution of 1 : 100,000 the lesions can be regarded as completely healed. In torpid lesions of the anterior segment of the eye as high a dilution of tuberculin as 1 : 1,000 can be resorted to, as only strong doses can produce a focal reaction. This limits the indication for tuberculin therapy.

In cases of metastatic tuberculosis of the eye tuberculin therapy gave the best results (87 per cent) in choroiditis. In keratitis and keratoscleritis the results were good in 65 per cent of the cases; in iritis and iridocyclitis the results were good only in 44 per cent.

In cases of allergic tuberculous disease of the eye favorable results were obtained in 37 per cent. This method is to be used only in addition to the general hygienic measures.

In metastatic tuberculosis of the eye tuberculin therapy is the basic method of direct specific action on the ocular process. It is a very favorable and effective method in the treatment of severe intra-ocular tuberculosis of the eye.

OLGA SITCHEVSKA.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

Eighty-Seventh Annual Meeting, Kansas City, Mo., May 13-15, 1936

DR. JOHN GREEN, ST. LOUIS, *Chairman*

DR. PARKER HEATH, DETROIT, *Secretary*

CHAIRMAN'S ADDRESS: THE PROMOTION OF BLINDNESS. DR. JOHN GREEN, ST. LOUIS.

Through individual research, but more often in collaboration with workers in the basic medical sciences, the ophthalmologist may hope to attain a knowledge of the causes underlying some intra-ocular maladies that today lead to blindness. The activities of the National Society for the Prevention of Blindness in conserving eyesight and preventing blindness are discussed. Attention is drawn to the large number of persons with unilateral blindness, or near blindness. This class is recruited largely from children with crossed eyes. Through improper advice leading to delay in securing expert advice and through inertia on the part of the parents, the crossed eye loses so much vision that it becomes useless from an economic standpoint. Unilateral blindness, or near blindness, is far more serious than it has actually been regarded.

The widespread incidence of trachoma in certain of the Midwestern States is discussed. Despite the valiant service of the United States Public Health Service in ferreting out and caring for the victims of this disease, the mass results are far from encouraging, and many are blinded.

The standards of ophthalmic practice are being raised to higher levels because of the ever increasing opportunities for systematic training and the work of the American Board of Ophthalmology. Each year there are fewer poorly trained ophthalmologists in practice. Those that remain are largely a product of "get equipped quick" institutions which are now universally discredited.

Much unnecessary blindness comes through chronic glaucoma. The factors to be considered are poorly trained ophthalmologists who either fail to make a diagnosis or allow the patient to drift into blindness without attempting either an energetic medical or surgical attack. Even when a correct diagnosis is made, it is, without social service workers, impossible to secure the complete cooperation of the patient to the end that medical treatment may be kept up unremittingly or surgical measures adopted before it is too late to retard the progress of the disease.

Attention is drawn to the frequent failure of physicians to diagnose intracranial neoplasms before irrevocable damage to sight has occurred as a result of the development and long continuance of papilledema. The tendency to use antisyphilitic remedies in the presence of a syndrome of brain tumor notwithstanding the absence of serologic or other evidence of syphilis is deplored. This period of useless and contraindicated therapy may be the precious time when an operation on the brain would save sight.

In search for focal infections or syphilis to account for certain inflammations of the cornea and uveal tract the ophthalmologist may forget the possibility that tuberculosis is the cause. Failure to recognize ocular tuberculosis may promote impairment of vision or blindness.

COMMON TUMORS OF THE EYELIDS. DR. C. S. O'BRIEN AND DR. A. E. BRALEY, IOWA CITY.

Among 100 consecutive tumors of eyelids, all of which were removed and studied under the microscope, there were 84 benign and 16 malignant neoplasms. The incidence of the benign tumors according to types was as follows: papillomas, 34; nevi, 17; sebaceous cysts, 13; fibromas, 5; sudoriferous cysts, 4; hemangiomas, 4; dermoid cysts, 2; molluscum contagiosum, 2; sweat gland adenoma, 1; xanthelama, 1, and granuloma, 1. The incidence of the malignant tumors was: carcinomas, 15, and malignant melanoma, 1. The clinical classification was found to be rather difficult at times; one cannot always make an accurate clinical diagnosis without microscopic studies.

DISCUSSION

DR. ARNOLD KNAPP, New York: Under the diagnosis of chalazion tumors of eyelids are operated on which are not chalazia, an error which can be rectified by the use of biopsy. In 1926 I saw a woman, 59 years of age, with a swelling of the upper lid, measuring about an inch by half an inch, not tender, definitely in the tarsus, not involving the overlying skin, with the conjunctival surface reddened, nodular and bulging, but with no glandular involvements. The tarsus and the tumor were removed from the inside of the lid just as in a tarsectomy. Except for a slight recurrence at the temporal extremity of the lid about four months later, the patient had no further trouble from the eyelid. The microscopic examination revealed carcinoma. A year later a swelling appeared in the preauricular region of that side. This was removed by a general surgeon; about nine months later a second operation was done with implantation of radium seeds. Slowly in the course of the next few years this malignant neoplasm descended down along the side of the face and of the neck, and ultimately the symptoms of an abdominal tumor developed. The latter condition caused the patient's death in 1933. This growth on the eyelid, if it had been seen early enough, would have been mistaken for a chalazion.

DR. ALGERNON B. REESE, New York: For comparison with Dr. O'Brien's group of tumors, I have listed 100 consecutive tumors removed from eyelids at the Eye Institute of New York City.

Epithelioma		28
Basal cell	21	
Squamous cell	4	
Basal-squamous cell	3	
Papilloma		18
Hemangioma		11
Nevus		8
Sebaceous cyst		6
Dermoid cyst		5
Sudoriferous cyst		5
Granuloma		4
Adenoma		3
Neurofibroma		3
Malignant melanoma		2
Xanthoma		2
Lymphangioma		1
Lymphosarcoma		1
Teratoid tumor		1
Foreign body reaction		1
Traumatic inclusion cyst		1

Epitheliomas were the most frequent, and two-thirds of them affected lower eyelids. Epithelioma involves the lower lid more frequently than the upper. The reason usually given for this predilection for the lower eyelid and the lower lip is that these are the areas which have the greater exposure to the actinic effect of the sun. Another possible reason is that these parts are subject more frequently to irritation. Certainly the lower lid is more frequently and more severely involved by blepharitis.

The list also shows that the basal cell is about five times more common than the squamous cell epithelioma. This is fortunate because, as Dr. O'Brien pointed out, the squamous cell type may metastasize, especially to the regional lymph nodes, whereas the basal cell type does not.

Three of the cases of granuloma were instances of what my associates and I for purposes of identification, call "novocainoma." Sometimes following an injection of novocaine (procaine hydrochloride) into the eyelids in preparation for a surgical procedure there occurs over the site of the injection a lump, the center of which shows necrotic tissue surrounded by granulation tissue and the presence of many foreign body giant cells. Various special stains disclose no evidence of bacteria or of any foreign substances. It is assumed, therefore, that the reaction is due to a chemical change. We have observed such lesions to occur a number of times, always in groups, and they cease to occur with the use of a fresher solution of the anesthetic.

ADENOCARCINOMA OF THE ORBIT. DR. WILLIAM L. BENEDICT, Rochester, Minn.

This article was published in full, with discussion, in the October, 1936, issue of the ARCHIVES, page 663.

MEDICAL AND SOCIAL ASPECTS OF CAUSES OF BLINDNESS IN PENNSYLVANIA. DR. ALFRED COWAN, Philadelphia.

An applicant for a pension for blindness in the state of Pennsylvania, to be eligible, is required, under the act providing for pensions for the

blind, to submit himself to examination from both the social and the ophthalmologic point of view. The latter investigation must be made by an accredited ophthalmologist. These requirements have made available for study and analysis nearly 6,000 reasonably descriptive reports. From the medical aspect the incidence of various ophthalmic diseases that were the sole or main contributing causes of blindness is chiefly considered in this communication. The causes of blindness are classified in groups as follows: congenital anomalies, traumas, diseases of the eyelids, conjunctiva, globe (glaucoma), cornea, uvea, lens, retina, vitreous and optic nerve, respectively, and miscellaneous. The social point of view has two principal concerns: (1) the education of the public to give prompt attention to any disease or injury of the eye and to any perceptible impairment of vision and (2) to lead the public to give attention to the lack of adequate medical facilities for the care of these conditions.

DISCUSSION

Dr. E. V. L. Brown, Chicago: The payment of goodly sums of money to the blind in the shape of pensions has again added to knowledge of the conditions which cause blindness, this time in an industrial and mining state, Pennsylvania. The only previous highly valuable report was from an agricultural state, Missouri. The unusual significance of these two reports is due to the fact that the laws in these states require an examination of the eyes by an accredited "eye physician." In Pennsylvania the information obtained by the local "eye doctor" is passed on to a state supervising examiner, Dr. Cowan, who has wisely interpreted the reports thus furnished him. He estimates that the present report, covering about 6,000 blind persons, represents about half of the number of blind persons in the state (11,852). The latter figure, when put alongside the 1930 Federal Census figure of 4,373 blind persons in Pennsylvania, demonstrates how effective is the inducement of money in bringing out an otherwise undisclosed condition. The Missouri law did much the same thing.

In these increasingly accurate statistics, some well known and some heretofore less well determined facts stand out. For instance, senile cataract was diagnosed in 22 per cent of those who became blind in Pennsylvania. Trauma caused 15 per cent of the blindness in this industrial state. The incidence of 17 per cent for uveal disease is surprisingly high. Ophthalmia neonatorum with 4 per cent shows what advance the work in prevention is making. Glaucoma rates high with 13 per cent, and congenital anomalies run to 5 per cent.

The ability of the state to deal correctly with blindness must, to a very important extent, be based on reliable information obtained by oculists before pensions are granted. It is therefore highly desirable, first, that other states follow the lead of Missouri, Colorado, Idaho, Oklahoma, Pennsylvania and the District of Columbia, where only an accredited ophthalmologist is allowed to determine whether or not a person is blind for the purpose of pensioning, and, second, that from data thus obtained a statistical analysis of the causes of blindness be reported.

DR. WILLIAM H. CRISP, Denver: The need for a definition of blindness arises largely out of the need for a standard on which to base the assignment of charity to those who because of loss of sight are prevented from gaining their livelihood. From the economic point of view, the standard of blindness is bound to vary with the individual patient. A former patient of mine, an old German, with vision of about 20/240, or less than 10 per cent of normal, was very successful in profitably raising young plants, which were bought by the farmers in his neighborhood. Yet another man with better vision than this will be quite unable to earn a living.

Statistics have been multiplied seemingly without end. Yet often the information afforded is far from reliable. Statistics are useful only as a basis for future action. Information about the blind in Pennsylvania is useful, for example, as indicated by Dr. Cowan, as a basis for the provision of various social services to the blind, including care of their general health, facilities for education and training, and opportunities for employment. Statistics have also an educational and prophylactic value in dealing with the general public. Unfortunately, there are some unreliable features of the information obtainable which can never be altogether remedied because of the remoteness of the time when the loss of vision occurred, the lack of technical records and often the inaccuracy in technical records because of carelessness or ignorance on the part of the general physician. There is no uniform system of tabulating diseases, and perhaps none is altogether desirable.

DR. CONRAD BERENS, New York: Although the group studied by the Committee on Statistics of the Blind is a fairly accurate cross-section of students in schools for the blind in the United States, the findings agree quite closely in certain respects with Dr. Cowan's statistics concerning blindness in the state of Pennsylvania. The committees' figures in regard to the inherited and congenital causes of blindness are naturally much higher (51 per cent) because the studies were concerned principally with young children. These studies clearly indicate the need for treating this group with greater intelligence. More thorough examinations and more carefully prepared records of the initial examinations of the patients will aid materially in obtaining the necessary statistical data regarding etiology.

DR. ALFRED COWAN, Philadelphia: My report contains only the data collected during the first year after the enactment of the law, and therefore it includes data on many persons who have been blind for any number of years. Data of this kind, of course, are not nearly so valuable as data obtained in future years will be, when reliable information about the causes in recently blinded persons will be available.

THE TRUE IMPORTANCE OF ANISEIKONIA. DR. EDWARD JACKSON, Denver.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

CATARACT FOLLOWING DINITROPHENOL TREATMENT FOR OBESITY. DR. WARREN D. HORNER, San Francisco.

This article was published in full, with discussion, in the September 1936 issue of the ARCHIVES, page 447.

A STATISTICAL STUDY OF MUSCLE FUNCTION TESTS IN AXIAL MYOPIA. DR. ALBERT C. SNELL, Rochester, N. Y.

In a tabulation of 1,078 consecutive cases of myopia, esophoria compared with exophoria was found to be present in the ratio of 2 to 1 up to the age of 40, and this ratio was not notably influenced by the degree of myopia or by the correcting glasses. The degree of exophoria and its relative frequency both increase moderately after 40.

Esophoria was present in 55.3 per cent, exophoria in 30.4 per cent, orthophoria in 9.6 per cent and simple hyperphoria in 4.7 per cent.

Convergence insufficiency is not a special characteristic of myopia. It was found in 1 per cent of the larger series and in 4 per cent of the smaller series of cases.

Myopes maintain their dynamic powers of convergence. The average amplitude of convergence in 90 per cent was found to be greater than the norm in 100 consecutive cases.

DISCUSSION

DR. BENNETT Y. ALVIS, St. Louis: Without careful measurements no such study could be made, and unless based on large numbers, covering considerable periods of time, such a study would be without significance. Dr. Snell's finding that esophoria and good convergence are more common among myopic patients than exophoria and weak convergence, also that muscle balance is generally more or less unchanged in most patients after the twelfth year, must therefore be accepted. I reviewed 41 records of myopia under 10 diopters in young myopic patients selected at random from my own practice:

	Myopes	Hyperopes
Average age	11.7 yr. (range, 4-35 yr.)	10 yr. (range, 4-24 yr.)
Time of observation	1 to 27 yr.	2 to 20 yr.
Average period	7.5 yr.	5 yr.
Myopic at start	37, or 90 per cent	.....
Hyperopic at start	.....	100 per cent
Myopia progressed in	32, or 84 per cent	.....
Hyperopia became less in	.....	7, or 22 per cent (none esophoric)
Maximum above	—5.00 diopters, 5	.....
Average maximum	—3.87 diopters	.....
Average increase	2.17 diopters	.....
Muscle balance:		
Orthophoric	21, or 50 per cent	11, or 44 per cent
Esophoric	11, or 27.5 per cent	11, or 44 per cent
Exophoric	9, or 22.5 per cent	3, or 12 per cent
Convergence near point:		
Cases in which it was recorded	33	27
Cases of convergent strabismus	1	5
Cases of divergent strabismus	1	..

Observations:	Myopia	Hyperopia
p'c = 1-2	inches (37 mm.) 55 per cent	p'c = 1-1½ inches (37 mm.) 68 per cent
p'c = 2	inches (50 min.) 30 per cent	p'c = 2-3 inches (75 mm.) 36 per cent
p'c = 3+	inches (75 mm.) 10 per cent	p'c = 5 inches (125 mm.) 4 per cent
p'c = 5	inches (125 mm.) 5 per cent	

On the basis of the cases presented here one may conclude only that there is no great difference in the proportion of esophorias to exophorias between young myopic and young hyperopic patients. One must surely agree with Dr. Snell that there is no evidence of inadequate convergence among myopic patients but rather a tendency to show convergence ability beyond the so-called normal. The question is of interest mainly in considering a possible causative relation between overconvergence and increasing myopia. The author, with the admirable reserve of the scientific attitude, refrains from drawing such a conclusion, but contents himself with showing that the convergence in myopia, both static and dynamic, is at least equal to that in other refractive states. There is no doubt that a preponderance of writers hold to the theory that convergence in near work is a principal causative factor in the increase of myopia.

Dr. Thomas D. Allen, Chicago: Muscle function tests are subjective. They vary decidedly from day to day and between two examiners. It is assumed, of course, that the author has carefully measured the interpupillary distance both for distance and for the near point and has carefully set the lenses to that measure. For if the interpupillary distance for distance is correct and the examination is made for near without resetting the lenses, one will have approximately a quarter of a prism diopter of esophoria for each diopter, owing to the difference of about 2.5 mm. in the pupillary distance. In a 12 diopter myope, this accounts for 3 prism diopters of esophoria.

Every one knows what axial myopia means, but how can one prove that it is present in the average myope up to 7 or 8 diopters? Dr. Dewey Katz in the paper which he read a year ago before the Association for Research in Ophthalmology explained how very difficult it was for one to be sure of the diagnosis of axial myopia. One cannot take the eye out and measure it or use a solution of iodine and roentgen rays in every case. All have seen myopic crescents or conus in some hyperopes and have been amazed not to find them in some myopes. It seems to me it would be better to drop the term or to substitute a term less likely to cause criticism.

Illuminating as is Dr. Snell's table on the near point of convergence as observed in 100 consecutive myopes, I feel that more information should be presented on such points as the relationship of the change in myopia to the change in esophoria and the near point of convergence. Dr. Alvis gave me one little idea about that in his discussions. Do persons with progressive myopia have a nearer point of convergence than persons with stationary myopia or persons with normal vision or with hyperopia? Certainly one must, in view of these findings, revise many conceptions one has and diligently reexamine one's methods of procedure. For if convergence increases myopia and if there is a tendency to esophoria in myopes, one should stimulate divergence, apply full corrections and encourage outdoor exercise.

Dr. Alfred Bielschowsky, Hanover, N. H.: Dr. Snell's results differ considerably from those which I obtained fifteen years ago in an investigation of myopia as an etiologic factor in a small but very interesting group of patients with convergent strabismus of a peculiar type. Patients with this type present the following characteristics: 1. The

convergent squint arises comparatively late, either in late childhood or in adult life. 2. Diplopia for distance is regularly the first symptom, in contrast to the other ordinary type of squint. 3. There is no diplopia for near except in inveterate cases. For near the homonymous double images are fused but appear again as crossed double images if the fixated object is brought closer. The behavior of the double images in the different parts of the field of fixation is very similar to that which is found in so-called divergence paralysis. 4. Another characteristic of the strabismus under discussion is that there is no excess of adduction as in typical convergence squint. On the other hand, only a slight deficiency of abduction is to be found. 5. In all my cases the peculiar disorder was attributed to physical or psychic exhaustion, the patients showing a neuropathic condition.

As to the pathogenesis, several etiologic factors play a part: (1) esophoria as the position of rest of the eyes; (2) weakness of the fusion apparatus; (3) increased tension of the medial recti, which is favored by an excessive approach to near work, which is partly due to the uncorrected myopia, partly to the esophoria, the overcoming of which is the easier the more the patient's eyes are approached to the fixated object, and (4) the presence sometimes, it must be assumed, of an increased tonus or spasm of the convergence innervation, which is rather frequently to be met with in functional neuroses.

As to the therapeutics, I must warn against adducting prisms (base out), which would support the increased tonus of the convergence innervation. Training of the power of abduction is sometimes useful, but fails more frequently. Bilateral advancement or shortening of the lateral recti will give the most gratifying results. Weakening operations on the medial recti are not advisable, because their function is not excessive.

CRITICAL ANALYSIS OF OPERATIONS FOR GLAUCOMA. DR. WEBB W. WEEKS, New York.

The object in this study is to determine as far as possible some of the factors primarily responsible for the unsuccessful control of increased ocular tension by the usual types of operative procedure. The following conclusions may be drawn:

1. The patient, unaware of visual disturbances or faulty vision and paying slight attention to discomforts in or about the eye, fails to report or come for operation at a stage of the disease favorable for successful operative intervention. Often prolonged local and general medical treatment allows pathologic changes to develop beyond the stage at which operation can aid in saving useful vision. Broadening the education of the general physician and the laity in the signs and symptoms of this disease is necessary.

2. The operative procedure chosen as the one best suited to relieve the pathologic conditions in a particular eye with persistent hypertension is, all too frequently, not the best one—for example, iridectomy for acute or chronic noncompensated glaucoma of some standing. A good history of the case and a thorough study of the conditions should lead to the choice of the proper operative procedure.

3. Frequently the operative technic has been faulty. In the hands of an astute, experienced and skilful operator this may not be true. It is decidedly a factor among those operating infrequently and among young men training in operative surgery. Faulty technic includes rough handling of the tissues, as seen in the free dissection of a conjunctival flap in trephine operations; using dull instruments, especially the trephine, keratome and scissors; improper making of a scleral or corneoscleral opening—too large, ragged and too small, too far forward in the cornea or too far posterior, injuring the ciliary body. Only training in the correct technic of the operative procedures to be used, making certain that the instruments are sharp and clean and frequent practicing on the eyes of cadavers or animals when dexterity and smoothness or instrumentation are uncertain will assure successful results.

4. Of the operative procedures now used in relieving hypertension in an eyeball in which the anterior segment has undergone pathologic changes, the filtering operations, i. e., the corneoscleral trephining of Elliott, the sclerectomy of Lagrange, the iridencleisis of Holth and the cyclodialysis of Heine, seem to have a definite use, the choice depending on the conditions found present in the eye to be operated on.

DR. HARRY S. GRADLE, Chicago: In all operations for glaucoma, the object of the surgeon is to restore the normal equilibrium of pressure. This equilibrium has a coarse balance, if one may use that term, in the form of a choroidal reservoir, and a finer balance in the aqueous humor. Among the few factors that the ophthalmic surgeon can influence in any eye with normal or abnormal tension the chief is the outlet of the aqueous. One cannot influence the total fluid content of the eyeball; one cannot influence the formation of the aqueous; one can influence only to a certain extent the elimination of the aqueous from the eye. There are three ways in which this can be done: One may through iridectomy increase elimination through the canal of Schlemm; one may open a new passage between the ciliary body and the sclera whereby the aqueous can filter into the perichoroidal spaces and there be absorbed, or one may form an extra-ocular drainage for the aqueous through a scleral opening into the subconjunctival spaces.

The type of operation that is to be employed must depend on the conditions found in the eye in question, and it is extremely difficult to lay down any set rules as to which operation should be performed. One does know that if firm anterior adhesions between the base of the iris and the posterior surface of the cornea have been formed one will find it a physical impossibility to open up the angle of the anterior chamber to restore the normal drainage of that eye. No spatula, no knife ever devised can free the adhesions in the angle so that the angle may again be opened to normal drainage. The formation of a new intra-ocular drainage path as exemplified in cyclodialysis is not difficult, but the application of that principle is limited to a small number of cases. It is obviously impossible for a pathway between the ciliary body and the sclera to remain open if there is excessive pressure on the other side of the ciliary body. In other words, if the pressure within the eyeball is high, it is entirely impossible for cyclodialysis to be successful. The final type of operation is that of extra-ocular drainage. Now, which type is one to employ, and why is one successful in some and not in others? Of

course, it stands to reason that the cases in which iridectomy may be used for opening up a normal drainage path are extremely limited in number; they include particularly cases of acute congestive glaucoma and those of some of the forms of secondary glaucoma. Again, in treating the chronic congestive glaucoma one knows that it is only with one of three types of operation that one can hope to be successful. The greatest difficulty lies in determining what operative procedure to use in treating the compensated forms of glaucoma. From the standpoint of choice, one would, of course, prefer to restore normal intra-ocular drainage in the suprachoroidal spaces when possible. In the failure of that, further intra-ocular drainage in the suprachoroidal spaces is preferable. An eye that has undergone a successful operation for extra-ocular drainage is a constant menace to the patient. There is always danger of hypotonia and of late infection, but in comparison with the dangers of the glaucoma itself those dangers have to be put to one side.

Dr. Philip D. O'Connor, Chicago: As Dr. Weeks has pointed out, in making the keratome incision the tendency is to go too far forward in the cornea or to enter the anterior chamber at such an angle that there is a flange of cornea posteriorly. In either case it is impossible to do a proper iridectomy. It seems to me that this cannot be emphasized too much. It seems that by making the incision with a knife, as in the Lagrange operation, these errors in technic could be avoided.

I should like to ask Dr. Weeks if he uses a subconjunctival injection of procaine hydrochloride before he makes the conjunctival flap in the trephine operation. I have made the flap with and without the injection, and I feel that the technic is easier and the results better when the subconjunctival injection is made. There is less trauma, more episcleral tissue is on the undersurface of the flap, the junction with the cornea is more clearly defined, and there is less danger in button-holing the flap.

Iridectomy with Cyclodialysis for the Reduction of Ocular Tension. Dr. John M. Wheeler, New York.

This article was published in full, with discussion, in the October 1936 issue of the Archives, page 569.

Early Ocular Complications of Epidemic Meningitis. Dr. Nathan K. Lazar, Chicago.

This paper will be published in full, with discussion, in a later issue of the Archives.

Effect of Intra-Ocular Concentration of Typhoid Antibody on Experimental Corneal Ulcers, with a Report of Seven Cases. Dr. Albert Louis Brown and Janet Pugh, Cincinnati.

This article was published in full, with discussion, in the September 1936 issue of the Archives, page 476.

MALFORMATIONS OF THE POSTERIOR SEGMENT OF THE HUMAN EYE: AN EMBRYOLOGIC INTERPRETATION. DR. BERTHA A. KLIEN, Chicago.

This article was published in full, with discussion, in the October 1936 issue of the ARCHIVES, page 624.

ON THE CORTICAL INNERVATION OF OCULAR MOVEMENTS. DR. NORMAN P. SCALA, Washington, D. C., and DR. ERNEST A. SPIEGEL, Philadelphia.

This paper is published in full, with discussion, in this issue of the ARCHIVES, page 967.

CLINICAL AND ANATOMIC OBSERVATIONS IN FELLOW EYES WITH CHRONIC TUBERCULOUS UVEITIS. DR. HARVEY D. LAMB, St. Louis.

Clinical and anatomic observations in right and left eyes of patients suffering from chronic tuberculous uveitis are described. The cornea was noted to be vascular. The angles of the iris contained a vast number of macrophages. The endothelia of the capillaries in the iris showed proliferation. The iris was thickened, and present in it were many plasma cells and macrophages. There were numerous posterior synechiae; also a partial pupillary membrane. Cyclitic lens changes were present. There was similar cellular infiltration of the ciliary body. Epithelial cells infiltrated the choroid, with many giant cells, plasma cells and macrophages. Some connective tissue changes were noted. The retina showed cystic degeneration and cellular infiltration. Many cells were seen applied to the surface of the papilla. The conditions were similar in the two eyes. While the changes were not all typically tuberculous, they could all result from tuberculosis.

DISCUSSION

DR. BEULAH CUSHMAN, Chicago: The specimens that come to the laboratory are those showing extreme pathologic changes, and these changes may be classified into the primary or the secondary type of tuberculous infection. Specimens showing primary infection show the forms usually found in children, with extensive coagulation necrosis, which go on to eventual perforation of the fibrous coat. The secondary infection is an allergic reaction with or without necrosis. In the allergic reaction there are extensive tubercle formation and cellular reaction with a minimum amount of necrosis, but the secondary types of infection in a nonallergic person may produce more extensive necrosis. These types of reactions vary according to the dose of bacilli and the resistance of the subject, and the variations of each type can be classified clinically into mild, moderate and severe. It is the severe forms, such as those shown by Dr. Lamb, which reach the laboratory.

In applying the help from the laboratory to the clinical problems, the experimental work of J. S. Friedenwald (Allergy and Immunity in Ocular Tuberculosis, ARCH. OPHTH. **9**: 165 [Feb.] 1933) helps and confirms the view from the histologic reactions of these various degrees of infection. He studied the reactions at different stages of tuberculous infection in normal guinea-pigs and in guinea-pigs which had been

desensitized with old tuberculin. The lesions found in these animals agreed with the clinical and pathologic observations in the different stages of tuberculous infection reported by different observers, and the results obtained seemed to substantiate the previous conclusion of Rich that the allergic reaction in tuberculous infection is a hindrance rather than a help to immunity of the host. Rich stated also that the allergy is certainly not uniformly distributed throughout all the tissues. The allergy in the tissues at the site of a tuberculous focus is far in excess of that in the rest of the body. Friedenwald classified the reactions of the tuberculous eyes of the guinea-pigs into three forms: first, the histologic changes with a certain dosage of tubercle bacilli in an animal which is immune and which also is allergic; second, the changes with a large dose of bacilli in an animal with immunity but no allergy—both secondary stages—and third, the changes with a large dose of bacilli in an animal with no immunity and no allergy—the primary infection.

When the results from these experiments are compared with the clinical findings one must agree with Friedenwald, Rich and many others that the aim of treatment with tuberculin should be the desensitization of the patient rather than the production of slight perifocal allergic reactions. In those patients who are not sensitive to tuberculin, one must depend on the general treatment to increase resistance and on local measures to stimulate fibrosis.

Dr. Lawrence T. Post, St. Louis: For the past three years my associates and I of the department of ophthalmology in Washington University have conducted a clinic for ocular tuberculosis. Fifty-two patients who presented definite evidence that their ocular lesions were tuberculous have been studied in great detail and followed carefully for long periods. Sixteen of these showed iritis. Three showed nodules. The patient described by the essayist was one of these. This woman was observed frequently by me and demonstrated to many students as having typical nodular tuberculous uveitis. There might be confusion with syphilis, sympathetic disease or leprosy, but these seemed definitely to be excluded. The most frequent condition which is probably usually tuberculous if syphilis can be excluded is the uveitis with gray translucent balls of loosely packed cells seen either in the substance of the iris or on its surface, frequently at the periphery or located on the endothelium of the cornea. Except for local therapy, attention to general health is by far the most important factor in the care of the tuberculous eye. Tuberculin therapy seems advantageous in some cases, but is certainly secondary to nutritious diet, rest and fresh air.

In spite of anything that it has been possible to do, the nodular type has almost invariably progressed unfavorably. In this particular woman almost every form of therapy was tried. She was watched particularly carefully because she happened to be taken into the employment in the home of one of the ophthalmologists on the staff. No treatment proved of any avail. There was a diagnosis of the possibility of an active tuberculous lesion, but there were no clinical signs of it. Even if the only systemic sign of tuberculosis is the roentgen sign, we refrain from giving tuberculin treatment. The first diagnosis from the sections of the second eye in this patient was sympathetic ophthalmia, for reasons

that the essayist has mentioned; hence the case was included in a series of 28 cases of sympathetic ophthalmia reported by me in 1933 at a meeting of the Southern Medical Association. Its inclusion was on purely histologic ground, because there was practically nothing suggestive of the condition except that. There had been two or three paracenteses of the eye, but that seems scarcely sufficient cause. On further study I believe that we are fully justified in considering that both eyes in this patient present tuberculous lesions.

TREATMENT OF STRABISMUS: INFLUENCE OF ORTHOPTIC TRAINING ON RESULTS OF OPERATION. DR. J. L. BRESSLER, Chicago.

This paper was published in full, with discussion, in the September, 1936, issue of the ARCHIVES, page 433.

Book Reviews

Die operative Therapie und die Pathogenese der Netzhautablösung. By Prof. Dr. Alfred Vogt. Stuttgart, Ferdinand Enke, 1936. Price, 65 marks. Pp. 174, with 238 illustrations.

Professor Vogt's treatise on the operative treatment and pathogenesis of detachment of the retina begins with a historical introduction which describes the older methods of treating retinal detachment up to the time of Gonin's epoch-making discovery of the importance of the retinal hole in the development of retinal detachment and of its closure by operation, with cure of the detachment.

Then follows a survey of the large amount of work on all phases of the problem of detachment, which has followed this discovery of Gonin, such as methods of localization of the hole in the retina and the various operations by ignipuncture, caustic procedures, diathermy, or electrolysis.

Vogt has given up surface coagulation, as the diffuse reaction clouds the picture in the fundus, so that a small remaining hole cannot be found at later operations. He prefers to treat only the opening in the retina and its immediate surroundings, thus avoiding unnecessary and sometimes dangerous injury to healthy portions of the retina, and he thinks that needle diathermy, needle catholysis and probably needle anode-electrolysis are the best methods. Catholysis has the advantage of aiding the ophthalmoscopic control and of requiring a much less expensive apparatus. The catholysis needle produces a glistening white foamy area, which is caused by the hydrogen and can be readily seen with the ophthalmoscope. Delicate punctate scars result without extensive changes of the surface. When the detachment is pronounced and the hole is far away from the surface of the affected area, it can be reached by the cathode needle. Vogt at first employed cathode-electrolysis for localization and then proceeded with the diathermy needle. Then he discovered that catholysis is sufficient to close the opening. He makes the puncture in one-half second with from 1 to 2 milliamperes current. The point on the sclera is marked with india ink and noted on the drawing. The next punctures are of longer duration (from one to three seconds) and greater intensity (3 milliamperes). If the hole is far distant, the needle can be introduced from 3 to 6 (or more) mm. to reach the retina; additional punctures are made surrounding the opening. This can be repeated in three days if the result is not satisfactory. The ball electrode is applied to the sclera not far from the puncture made with the needle. Hemorrhages do not occur any more frequently than by any other method of puncture, and late hemorrhages have not been observed.

Vogt is not convinced of the value of the stenopaic spectacles, as he believes that succussion of the vitreous takes place in opening and closing the lids and that this is not influenced by wearing the stenopaic glasses.

If the retina is so far detached that it masks the point of puncture, Vogt uses a 0.2 mm. catholysis needle which is 3, 4 or 6 mm. long, so that the surface of the retina can be reached; then punctures are made with diathermy needles from 1 to 2 mm. long until enough fluid escapes to allow the retina to become replaced.

As to prognosis Vogt says that any recent detachment (not older than three or four months) can be operated on with some prospect of success. Diathermy punctures give better prospect, especially if the needles are fine and sharp and the applications to the margins of the hole are correctly applied. The diagnostic cathode puncture must be applied for the briefest period and should produce little foam.

In discussing the causes of detachment the author finds no difference between myopic and senile detachments, as both depend on peripheral degeneration of the fundus. Vogt denies the rôle of the vitreous in the production of detachment, particularly because the slit lamp has not shown a fixed band in the vitreous but rather a greater motility of the vitreous structure than normal. Furthermore, the multiple appearance of retinal tears is difficult to explain by the Leber-Gonin retraction theory. Instances of detachment of the vitreous occur frequently in senile and highly myopic eyes without retinal detachment and are a part of senile and myopic degenerative changes which occur in senile and myopic eyes.

There are several types of retinal holes. They represent destructive processes leading to decay of the retina accompanied by vascular occlusion, just as changes are observed in the vitreous, choroid and marginal vascular network. The variety of tears, their location and difficulty of diagnosis (hemorrhage) are next treated. The author describes ophthalmoscopic changes which he reported in 1930, situated in the periphery, consisting of delicate branching netlike white lines, which enclose red translucent thin-walled spaces. These he regards as cystoid changes, and these spaces can become holes or tears. The relation of the white lines to obliterated vessels is unsettled.

Vogt believes that cathode electrolysis has a special value in the treatment of macular holes, as a number of fine punctures can be made surrounding the macula which reattach the retina in this area.

Recent cases of retinal detachment have been examined histologically. These cases were reported by Kümmel, Sourdille, Redslob and Poulard-Veil and show that no inflammatory changes are present in the vitreous and that all the changes are present in the retina alone.

Vogt emphasizes that vital changes in the retina itself are responsible for detachment instead of lesions in the choroid and vitreous. Cystoid degeneration and atrophy of the retina are the vital and destructive factors which finally produce the extreme friability of the retina and spontaneous formation of holes. This process has been confirmed by the histologic examination of recent retinal detachments and has entirely changed the views of ophthalmologists as to the cause of detachment of the retina. This degeneration is connected with vascular disturbances and resulting nutritional changes.

This book is characterized by the excellence and beauty of the many drawings in color, which are so lifelike and instructive that a

mere study of the pictures will be of great benefit to the ophthalmologist.

Professor Vogt has brought out many interesting points in this treatise on retinal detachment, and his views on the causation of retinal holes will be sure to stimulate study and research. Thanks are also due him for drawing attention to the good points of cathode electrolysis, its simplicity and the cheapness of the apparatus, though it is not clear whether in his opinion it can replace diathermy.

ARNOLD KNAPP.

On Deficiency of A Vitamin and Visual Dysaptation. By Dr. C. Edmund and Dr. S. Clemmesen, Copenhagen. Price, 5 kronen. Pp. 92, with 8 diagrams in the text. Copenhagen: Levin & Munksgaard, and London: Humphrey Milford, Oxford University Press, 1936.

Edmund and Clemmesen have devised an accurate clinical test for determining and recording the degree of visual disturbance present in persons who are suffering from a deficiency in the adaptation of the eyes to reduced illumination. Years ago Edmund used for these tests a white Masson disk on which were drawn eight black sectors of different sizes. When the disk was rotated there appeared eight gray rings of different shades ranging from a nearly white tone to one almost black. The *power of distinction,* as he calls it, could then be determined by finding the palest gray ring which the observer could distinguish from the white ground. For clinical use this test was modified by the authors, and large test letters of different gray tones on a white ground were substituted for the gray rings. These letters were viewed through closely fitting test frames, excluding side light, in which were placed in succession the ten different gray photometric glasses devised by Tscherning. Thus the gray letters were seen under ten grades of reduced illumination, and the power of distinction was measured for the different illuminations after a ten minute period of adaptation of the eye to each. The values obtained for the subject tested were then plotted in a curve, a coordinate system being used in which the divisions on the ordinate axis indicated the power of distinction, while the divisions on the abscissa axis indicated the degree of illumination. Considerable space is devoted to the technical execution of the tests, the mathematical computation of the values obtained and the discussion of dysaptation in general.

It was found that the healthy eye attains its maximum power of distinction only within a limited range of illumination. With increased illumination vision suffers because of dazzling, and as illumination is diminished vision decreases accordingly—a physiologic hemeralopia. Moreover, for any given illumination the best vision is obtained only after a few minutes of adaptation. In hemeralopia, of whatever variety or origin, vision is greatly reduced as illumination decreases, and the best vision lies always in a zone of illumination somewhat lower than that in which the healthy eye functions best. And in the degree of illumination best for the healthy eye dazzling comes on in the hemeralopic eye. Nyctalopia, the condition characterized by dazzling and decreased vision in normal illumination, was found to exist in every case of

hemeralopia. Hence the authors believe that the term nyctalopia may be discarded and the term dysaptation used perferably for both the nyctalopic and the hemeralopic state.

The tests were carried out on normal persons, on those with congenital hemeralopia, on those with hemeralopia resulting from retinitis pigmentosa and glaucoma, and on pregnant women in the hospital with complications such as excessive vomiting and nephritis. In this group of pregnant women 50 per cent exhibited a disturbance of adaptation, and this dysaptation was cured in every case by the administration of vitamin A orally or parenterally. With pregnancy and diseases of the eye excluded, dysaptation was found in but from 5 to 6 per cent of ordinary female patients in hospitals. The examination of thirty-nine hospital nurses, as a check, revealed no cases of dysaptation, although in March the administration of vitamin A raised the lower limit of the normal zone of best visual perception to the upper limit. This suggested a deficiency of vitamins in the nurses' diet.

This instructive volume concludes with a twenty-two page reprint of a recently published magazine article in which the subject of dysaptation is discussed further and a number of histories of cases are recorded.

WARD A. HOLDEN.

Squint Training. By M. A. Pugh. Price, $2.75. Pp. 117, with illustrations. London: Oxford University Press, 1936.

This book is a first-class guide to the nonsurgical management of squint and should become the textbook for all those who desire to cope with the problem intelligently. The author, Mary A Pugh, medical officer in charge of the orthoptic department at the Royal London Ophthalmic Hospital, is, because of intensive experience with a large amount of clinical material, as well qualified to write on the subject of orthoptic training as any present day ophthalmologist.

To quote from the preface, "The academic side of strabismus is not dealt with in this book, which aims only at giving a practical classification of cases, an accurate method of measuring the deformity, and the technique of treating a squint."

To quote further, "Stereoscopes of any design are not safe to order unless the patient is very intelligent or has developed normal projection. Careless exercises with this instrument may fix a squint instead of training the eye to straightness."

American ophthalmologists will find themselves at a disadvantage in utilizing all that this book has to offer, because of the lack of an American-made, reasonably priced major amblyoscope of the orthoptoscope type and an adequate flashing device. Proper technic demands an instrument which can be accurately adjusted to the true visual axes of the two eyes, regardless of whether the deviation be horizontal, vertical or cyclotorsional, and a flasher which will stimulate the two maculae alternately at speeds varying from one to twenty times per second on each slide.

The importance of abnormal retinal correspondence is emphasized by the statement that it was present in about 50 per cent of the patients

treated. Although this condition is a difficulty which must be surmounted before training to decrease the angle of deviation can be started, the author states that an intelligent adult, by means of occlusion and exercise, may overcome the disability. Its presence will, of course, result in an attempted reestablishment of the old deviation following surgical intervention and defeat any efforts to stimulate normal binocular vision by training. Complete and continuous occlusion of the good eye must be practiced until the visual acuity has become at least 0.4 and normal projection has been established. Exercise may then be started under careful supervision with a major amblyoscope to stimulate binocular perception and, later, fusion amplitude.

About the somewhat discouraging subject of alternating squint the following statement is made: "Alternating squints have been regarded with unnecessary pessimism since the percentage of cases which will not develop fusion is small. On the other hand, it is doubtful whether the end-result is worth the trouble. The patient must decide. Operated alternating squints frequently develop a new abnormal correspondence on the basis of a smaller angle of deviation."

The various squint-training devices in use in England are described and the fundamental principles of their use, with an estimate of their value, are given.

In a series of three hundred consecutive patients showing normal retinal correspondence and normal projection, 38 per cent had straight eyes with good fusion and, in most cases, good stereoscopic vision after orthoptic treatment alone. Pessimism is expressed regarding the ability of glasses alone to straighten squints unless the patient is seen immediately after the onset of the deviation. Operation is resorted to when training fails. Statistics are given for cases classified under the following heads: (1) refraction alone; (2) refraction and occlusion; (3) orthoptic training; (4) refraction, occlusion, operation and orthoptic training. The various statistics are analyzed and discussed.

The book is too full of material for an abstract to do it justice.

K. C. BRANDENBURG.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St. Honoré, Paris.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Holland.
Place: Cairo. Time: December 1937.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35, Harley St., London, W. 1.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

German Ophthalmological Society

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. William L. Benedict, 102 Second Ave., S. W., Rochester, Minn.
Secretary: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Place: Atlantic City. Time: June 7-11, 1937.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City, Utah. Time: May 24-27, 1937.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.
Place: Seattle. Time: January 1937.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. A. H. Pember, 500 W. Milwaukee St., Janesville, Wis.
Secretary-Treasurer: Dr. W. H. Elmer, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Leslie R. Hazlett, 100 S. Main St., Butler.
Secretary-Treasurer: Dr. C. W. Beals, 41 N. Brady St., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227, 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hosp., Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. John F. Fairbain, 925 Delaware Ave., Buffalo.
Secretary: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. Burton W. Fassett, Geer Bldg., Durham.
Secretary-Treasurer: Dr. Casper W. Jennings, 332 N. Elm St., Greensboro.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Trygve Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 514 6th St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1936.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Emory Hill, 501 E. Franklin St., Richmond.
Secretary-Treasurer: Dr. George G. Hankins, Medical Arts Bldg., Newport News.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. B. E. Failing, 31 Lincoln Park, Newark, N. J.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. William O. Martin Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. A. V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. Robert B. Blue, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. F. W. Thomas, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. H. D. Emswiler, 370 E. Town St., Columbus, Ohio.
Place: Athletic Club. Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. T. L. Goodman, 602 W. 10th St., Forth Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. William A. Boyce, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Hilmar G. Martin, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer. Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member in alphabetical order.
Secretary: Dr. C. W. Rucker, 527 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. W. J. Blackburn, 663 Reibold Bldg., Dayton, O.
Secretary-Treasurer: Dr. F. J. Driscoll, 986 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Kate S. Zerfoss, 119, 7th Ave., N., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LaGrand H. Hardy, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. E. C. Reynolds, 657 Main Ave., Passaic, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 80 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Robert H. Courtney, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Carl T. Eber, 308 N. 6th St., St. Louis.
Secretary: Dr. W. M. James, 508 N. Grand Ave., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Lewis F. Morrison, 490 Post St., San Francisco.
Secretary: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Greene, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto.
Secretary: Dr. A. Lloyd Morgan, 86 Bloor St., West, Toronto.
Time: First Monday, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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